LXX.

CONTRIBUTION TO THE PATHOLOGY OF THE LABYRINTH.*

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MR. PRESIDENT AND GENTLEMEN:

Before entering on the subject of labyrinthine pathology, I should like to say a few words about the method by which the inner ear may be microscopically examined. Although this method is a tedious and often disappointing one, I hope that some of the younger otologists in this country may be induced to take it up. There are still large gaps in our knowledge regarding the pathology of the labyrinth. For instance, no one has yet, as far I know, described the microscopic pathology of deafness due to mumps. Neuritis of the eighth nerve following infectious fevers has not been fully elucidated. The pathology of old age deafness is still in dispute—some observers ascribing it to degenerative changes in the cochlear gang-

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lion cells and in Corti's organ, others to calcification of the basilar membrane. The changes in the inner ear in congenital and acquired syphilis have not been fully explained on a pathologic basis, nor has the neuritis which is so commonly observed in cases of toxemia from various causes. I could add to this list, but I have said enough to demonstrate the need for further investigation. I earnestly hope that American otologists may soon be able to fill in the gaps in our knowledge.

1. THE MICROSCOPIC EXAMINATION OF THE MIDDLE AND INNER EAR BY THE CELLOIDIN METHOD.

Preparation of the Block of Bone Containing the Middle and Inner Ear.—The temporal bone must be obtained as soon as possible after death—certainly within twenty-four hours. In removing the brain the seventh and eighth nerves should be cut close to the pons, so as to leave as much as possible attached to the temporal bone. The dura mater is cut with a knife round the internal auditory meatus in order that the nerves may not be pulled out when the dura is stripped off. The saccus endolymphaticus is examined with the naked eye, and for this purpose a crucial incision is made into it. ·The dura mater is now removed with forceps from the upper and posterior surfaces of the petrous bone.

With a small butcher's saw a cuboid block containing the important parts of the middle and inner ear is cut from the temporal bone. The first saw cut is made in a vertical direction at right angles to the long axis of the petrous pyramid just in front of the internal auditory meatus. The second cut is made parallel to and behind the first, through the mastoid antrum, posterior to the semicircular canals. The third vertical saw cut is made in an anteroposterior direction through the middle cranial fossa and external auditory meatus and runs parallel to the posterior surface of the petrous pyramid and to the middle ear cleft. The last cut runs through the jugular fossa and is made in a horizontal direction below the middle and inner ear.

In this way a six sided block is obtained, but it requires some trimming. The anterior wall of the external meatus should be cut away with bone forceps, so that the tympanic

membrane may be exposed and its condition noted. The jugular bulb is also inspected and thereafter its lining membrane is removed with dissecting forceps. The carotid canal is opened up with bone forceps and the artery removed—care being taken not to injure the eustachian tube, which lies just external to this vessel. Finally, the superior semicircular canal is opened with bone forceps in order to allow the fixing fluid to gain access to the inner ear. (It must be noted that the superior canal lies somewhat in front of the arcuate eminence.) The block so obtained is washed in running water to remove bone dust and is then placed in a wide mouthed glass stoppered bottle containing 5 per cent formol or Jore's fluid for one month—the fluid being changed on several occasions. If the nerve structures are to be specially examined, it is best to place the block in Müller's fluid after it has been twenty-four hours in formol. The Müller's fluid must be changed frequently.

Decalcification, Washing and Hardening.—After the block has been fixed it may be decalcified in 5 per cent formol and 5 per cent acid (equal parts) for one month or six weeks, according to the size of the block. (Perenny's solution may be used instead—10 per cent nitric acid, 364 c. c.; methylated spirit, 336 c. c.; chromic acid, ½ per cent, 300 c. c.) During the first week the fluid is changed daily, during the second week every second day, and during the third and following weeks the fluid should be changed twice weekly. After decalcification the block is gently removed and placed in a porcelain dish and washed in running water for four or five days. The specimen is next hardened for the second time, first in 70 per cent spirit for twenty-four hours, then 80 per cent for twenty-four hours, then methylated spirit for three days (this latter solution being changed daily). From methylated spirit the block is passed into absolute alcohol for two days (changed daily), and then into absolute alcohol and ether (equal parts) for two days (changed daily). During the hardening process it is convenient to use a series of ten or twelve wide mouthed glass stoppered bottles.

Embedding.—The block is now put into thin celloidin for one month—a bottle similar to the above being used—and then transferred to thick celloidin for one month. (During the

time the block is in the thin celloidin it may be an advantage
to employ an evacuation pump so as to get rid of air bubbles
from the hollow spaces of the middle and inner ear, but the
negative pressure employed must not be excessive, otherwise
the structures of the membranous labyrinth may be distorted.
It is better to reserve the evacuation pump for cases in which
one finds on microscopic examination that the hollow spaces
of the ear contain air cells. The specimen should then be put
back in absolute alcohol and ether for six hours and then
transferred to thin celloidin before the pump is employed.) At
the end of the second month of embedding, the lid of the glass
jar containing the block is left partially open so that the speci-
men may gradually dry and harden. When the celloidin is
thoroughly hardened—usually in about a week—the block is
cut out with a knife and pared down with a razor to the re-
quired size—i. e., a cube measuring about three-quarters of
an inch in all dimensions.

Cutting.—The block may be cut in one of two directions:
(1) Horizontally, from above downwards; in this way some
400 sections are obtained, 20 micromillimeters in thickness. (2)
The specimen may be cut vertically, from before backwards;
in this way about 700 sections are obtained. After deciding
in which direction the block is to be cut, it must be fixed on
a piece of stabilit; the block is dipped into absolute alcohol
and ether for a moment and then into thick celloidin and
placed on the stabilit with the surface to be cut uppermost.
The specimen should now be left for half an hour or more in
the air so that the celloidin may harden and then placed in
a bottle of methylated spirit for six hours. The stabilit block
with the specimen attached is then removed and fixed in the
Schantze microtome. If the block is very hard sections may
sometimes be obtained 10 mm. in thickness, but as a rule 20
mm. give sufficiently thin sections. The writer has been in
the habit of numbering and staining every fifth section only;
the intervening ones are placed in a large fruit jar containing
spirit. In very important cases, however, such as deafmutism
or otosclerosis, every section should be numbered and stained.
In less important cases the sections which are selected for
staining are placed on a slide and wrapped up in a strip of
blotting paper moistened with spirit; they must on no account

be allowed to dry.   Before the blotting paper is put on each slide is numbered with a diamond, thus—"William Smith, 5, 10, 15, etc."   As each hundred sections are cut, the selected twenty are tied up together in a bundle, labeled and kept moist with spirit.   When all the sections have been cut they are placed in a large glass jar containing spirit.   Staining may be deferred to a convenient occasion.

Staining.—The blotting paper is unwrapped and the section removed by tapping the slide against the side of a bowl containing water.   The section is now lifted with a copper section lifter and placed in a watch glass containing filtered hemalum (Mayer's).   In this the section remains for a period varying from five to thirty minutes, according to the quality of the staining solution.   The section is then lifted out and thoroughly washed in a bowl of water, and then transferred to a watery solution of eosin ($\frac{1}{4}$ to $\frac{1}{2}$ per cent).   The time taken for staining with eosin varies from two to ten seconds as a rule. The specimen is again washed in a second bowl of water and transferred to a third watch glass containing spirit, in order to remove the excess of eosin.   From this it is passed on to a fourth and fifth watch glass containing spirit, and is then placed for a second or two in a sixth watch glass containing absolute alcohol.   Finally, the specimen is removed to a seventh glass which holds carbol-xylol, where it remains for a minute or two until it sinks to the bottom of the glass.   The section is then transferred with the aid of a needle and a copper section lifter to the numbered slide from which it was taken for staining purposes.   It is now covered with Canada balsam, and with the aid of a needle a clean cover slip is slowly let down over it so as to exclude air bubbles.

Other staining methods may be employed—e. g., van Giesen, iron, alum, osmic acid, etc.   For details of these methods the reader is referred to textbooks on histology.

It is possible to work three rows of watch glasses (twenty-one in all) containing the various solutions at one time, and in this way from sixteen to twenty sections can be stained in two hours.

It will be seen that the process required for the microscopic examination of the middle and inner ears is a somewhat lengthy one, taking as it does about six months.   It must also

be confessed that many difficulties and disappointments are met with, notably injury to the ear in cutting out the block of bone, failure to decalcify sufficiently, presence of air cells in the hollow spaces of the middle and inner ear, and faulty staining. For all these reasons the microscopic examination of the ear is not a task to be lightly undertaken.

2. ANATOMY OF THE LABYRINTH.

It is not my intention to inflict on the society an account of the anatomy of the inner ear. It may be, however, that some of the members are not accustomed to examine micro-scopic sections of the labyrinth and consequently may find it difficult to recognize the various parts of the membranous labyrinth as they appear in sections. For this reason I propose to show a series of photomicrographs of the inner ear (of very low magnification) as it appears when cut (1) in vertical sec-tions from before backwards, and (2) in horizontal sections from above downwards. (Figs. 1 to 16.)

3. INJURIES.—FRACTURE.

Fractures of the cranial base frequently involve the middle and inner ear. (There is disturbance of hearing in 24 per cent of the cases of basal fractures who recover.) The line of fracture may run parallel to the long axis of the petrous bone or at right angles to the long axis.

(1) Longitudinal fractures start in the region of the sella turcica, and pass backwards along the line of the middle ear cleft, breaking the roof of the eustachian tube and tympanic cavity. The fracture may then pass outwards to the external meatus and squamous region; if this is the case the inner ear is not involved, though the ossicles may be dislocated and the drumhead torn. On the other hand, the fracture after reach-ing the roof of the tympanic cavity may pass inwards through the petrous pyramid and thus resemble those fractures which run at right angles to the long axis of the petrous bone. The inner ear is of course involved in these latter cases.

(2) Transverse fractures of the petrous pyramid always injure the labyrinth. As a rule the fracture passes through the external meatus, roof of the tympanic cavity, vestibule and internal auditory meatus, as this is the line of least resist-ance. The fracture may, however, pass further forward

through the cochlea, or further back when the canals are involved.

If the patient survives the injury at the time, he is not out of danger, as meningitis may supervene as the result of (1) infection from the eustachian tube and middle air spaces, or of (2) contamination of the extravasated blood via the external meatus and tympanic cavity. Meningitis is of course more likely if the labyrinth is involved in the fracture.

Cases of labyrinth injury associated with fracture of the base may be divided into (1) recent and (2) old. (1) In the former cases we have marked hemorrhage into the inner ear. Even in those cases in which the labyrinth capsule is not injured we meet with small hemorrhages in the inner ear and eighth nerve. The auditory nerve may be ruptured and the crura of the stapes torn from the footplate. (2) In old cases the line of fracture may be visible—partly closed by fibrous tissue and partly by new bone. In a case examined by Nager many years after injury there was new connective tissue and bone in the vestibule and in the basal coil of the cochlea. In other cases the line of fracture is not evident.

Case 1.—Male, aged 54, knocked down by a street car. On admission the patient was pale, irritable and restless; pulse 64, temperature 96° F. Bruises were present on the back of the scalp and above the left ear. There was profuse bleeding from the right ear, but no flow of cerebrospinal fluid. (As will be seen later the labyrinth capsule in this case was not injured.) The right pupil was more dilated than the left. Four hours later the patient became comatose and the breathing stertorous. The left arm was found to be flaccid. Operation was performed by a general surgeon. On incising the dura mater blood escaped. The patient died ten hours later.

Postmortem.—The fracture runs along the roof of the middle ear cleft. The middle meningeal artery is lacerated and the roof of the tympanic cavity is splintered. The tympanum and mastoid cells are filled with blood. The fracture extends from the anterior part of the left middle fossa obliquely backwards through the squamous temporal and parietal bones to the lambda, and thence to the right parietal and squamous regions. .From here it passes down to a point behind the

right petrous temporal, crosses the sigmoid sinus and then runs along the anterior border of the right petrous bone.

Microscopic Examination of the Right Ear.—The tympanic membrane is ruptured; blood present in tympanum (Figs. 17 and 18); hemorrhage in the region of the geniculate ganglion (Fig. 19); bleeding in the fossa subarcuata. The cochlea shows slight hemorrhages in the modiolus (Fig. 20), but the scalæ of the cochlea are free from blood (Fig. 17). The vestibule and canals show no extravasated blood, but hemorrhage is present in the fundus of the internal meatus (Fig. 19) and in the aqueduct of the cochlea.

Case 2.—Male, aged 41, fell from a scaffold only nine feet high. When found the patient was conscious but was bleeding from the right ear. On admission the face was pale and pulse feeble. There was a flow of blood and cerebrospinal fluid from the right ear. (Microscopic examination showed fracture of the petrous bone involving the labyrinth and extending from the inner wall of the middle ear to the internal auditory meatus.) The patient was very deaf. Coma supervened and the breathing became stertorous. Later general convulsions came on, the eyes being turned to the right— i. e., conjugate deviation to the side of the labyrinth lesion. The right pupil was dilated. Left side of the face and left arm became paralyzed, and the respiration of the Cheyne-Stokes type; pulse 76. An operation was performed by a general surgeon. A fissured fracture was found running from the anterior part of the right squamous temporal downwards and backwards. The bleeding point could not be located. The external carotid was therefore ligatured. The patient died eight hours after operation.

Postmortem.—Skull thicker than normal; carotid arteries thickened; considerable amount of clot present behind the trephine opening. The brain had not expanded. Both lungs showed several gummata. The fracture bifurcated, one limb passing along the roof of the middle ear cleft and the other passing inwards through the labyrinth. In preparing the specimen the outer wall of the middle ear, along with the malleus, incus and tympanic membrane became separated, as they were already loose. Blood was present in the eustachian tube, tympanic cavity, mastoid antrum and cells.

Microscopic Examination of the Right Ear.—The line of fracture passes through the superior and external canal (Fig. 21), downwards through the oval window (Fig. 22), involving the stapes, and below this through the promontory (Fig. 23). The fracture reaches the internal meatus (Fig. 23), but does not extend to the dura covering the posterior surface of the petrous bone. Posterior crus of the stapes is broken. Cochlea: There is marked hemorrhage in the spiral ligament and in the scala tympani and vestibuli, especially in the basal coil (Figs. 22 and 23). There is very little blood in the cochlear canal itself. There are a few small scattered hemorrhages in the modiolus (Fig. 22). The utricle and saccule are ruptured and there is blood in both the peri- and endo-lymphatic spaces of the vestibule (Fig. 22). Semicircular canals: Blood is present in the endolymphatic and perilymph spaces of the superior and external canals (Fig. 21). There is slight hemorrhage along the vestibular nerve, especially along the branch to the saccule. The cochlear nerve to the basal coil also shows hemorrhage (Fig. 22). There is comparatively little hemorrhage present in the internal meatus.

Case 3.—Male, aged 44, laborer. Two days before admission, as he was going home under the influence of alcohol, he slipped and fell on the pavement, striking the right side of his head. He was not unconscious after the accident, but there was bleeding from the right ear and also from the mouth. After the accident the patient suffered from severe vertical headache. On admission he felt as if he were rotating from left to right about a vertical axis. The right ear has been quite deaf since the accident. He has felt sick but has not vomited.

Examination.—Watery discharge from right ear. Right tympanic membrane red and bulging; complete deafness in right ear: Weber lateralized to good ear; spontaneous nystagmus to left (sound side) of second degree; patient tends to fall to the right and shows a pointing error to the right; no facial paralysis. Temperature, 101° F., rose to 103° F. Kernig's sign absent; lumbar puncture: cerebrospinal fluid blood stained but not under pressure. Two days later patient very ill. Severe headache and backache. Temperature 105°, pulse only 72; Kernig's sign present; slight facial paralysis on right

side; second lumbar puncture; fluid under great tension. Poly-morphs greatly increased.

Postmortem.—Blood present over the vertex in subdural space and in right temporal region. At the base purulent lepto-meningitis was present over pons, medulla and lower surface of cerebellum. Laceration of left temporosphenoidal lobe. The right temporal bone showed a stellate fracture involving both petrous and squamous portions.

Microscopic Examination. External Auditory Meatus.— The anterior wall is fractured (Figs. 25, 26 and 27). The tympanic membrane is generally thickened and is ruptured be-low and behind the handle of the malleus (Fig. 27). The outer wall of the eustachian tube is fractured. There is a fracture through the roof of the tympanic cavity (Figs. 26 and 27). There is also a fracture of the floor of the tympanum—i. e., of the roof of the jugular bulb (Fig. 25). There is hemorrhage in the outer part of the attic (Fig. 26). In the lower part of the tympanum posteriorly the exudate is purulent (Fig. 26). The stapes shows at least two fractures. The malleus and incus are not fractured. The fracture goes through the facial canal above the oval window and passes through the upper margin of the window. There is also a fracture of the region of the round window (Fig. 25). One can trace the continuity of the exudate in the niche of the round window with that in the scala tympani. Cochlea: Only the intravestibular portion of the cochlea is fractured (Fig. 25). There is hemorrhage in the spiral ligament. Corti's organ is unrecognizable. All three scalæ contain exudate, but the scala tympani shows much more than the others (Fig. 24). Vestibule: The fracture runs into the roof of the vestibule and then downwards through the bony spiral lamina and the region of the round window into the tympanic cavity and finally passes through the floor of the cavity (Fig. 25). The vestibule itself is almost com-pletely filled with hemorrhage. The tip of the promontory is almost chipped off. Canals: There is no fracture of the labyrinth capsule in the region of the canals. The lateral canal contains pus and blood (Fig. 26). The other canals are more normal. Internal meatus: The fracture extends right through from the floor of the middle fossa, through the bony roof and floor of the meatus (Fig. 24), to the region of the opening

of the aqueduct of the cochlea. Some cellular exudate is present in the fundus of the internal meatus (Fig. 24).

Case 4.—Male, aged 6, suffered from fracture of the base of the skull in August, 1913, with bleeding from the right ear. He recovered and remained well for one year, till August 30, 1914, when earache (right ear) and headache came on. August 31, 1914: vomiting, unconsciousness, jerkings of right arm. Rigidity of neck. Kernig doubtful. Temperature 101° F., pulse 110. Death September 1, 1914.

Postmortem (five and a half hours after death).—Flattening of convolutions; cerebrospinal fluid increased and turbid; exudate in interpeduncular space. Sphenoid and ethmoid healthy. Left middle ear contained turbid fluid. Old fracture of roof of right mastoid antrum.

Microscopic Examination of Right Ear.—The mastoid cells contain pus (Fig. 29). There is a gap in the roof of the mastoid antrum filled with fibrous tissue, and there is a piece of bone lying loose in the midst of this fibrous tissue, with pus around it (Fig. 29). This piece of bone proves to be the incus, which had become dislocated backwards into the mastoid antrum. From the appearance of the well formed fibrous tissue surrounding the incus it is evident that this is an old dislocation. (What appears to have happened is that the incus became dislocated backwards at the time of the fracture in 1913, retaining its attachment to the floor of the aditus, and at the same time there was a fracture of the roof of the antrum. The patient recovered at the time, as no infection occurred, but one year later when he developed an acute suppurative otitis media the infection passed by way of the tympanic cavity, the aditus and antrum, through the old gap, which was apparently filled with fibrous tissue, to the intracranial structures, and the result was purulent leptomeningitis and death.) The lining of the eustachian tube is very vascular and swollen, and the submucosa is infiltrated with pus cells. The tympanic cavity contains pus. The footplate of the stapes is quite normal. It is the long process of the incus which has been broken (Fig. 29). The head of the malleus is ankylosed to the new bone which has been formed in the roof of the middle ear (Fig. 28). Cochlea: There are many white cells in the scala tympani in the basal coil, but the pus does not extend as far

as the helicotrema. There is some infiltration of pus cells from the internal meatus into the spiral ganglion of the basal coil. Corti's organ is quite normal. The vestibule and canals are quite healthy. Internal meatus: There are many white cells in the internal meatus within the arachnoid sheath.

4. WAR INJURIES OF THE EAR.

A description of war injuries of the ear written by a surgeon at a clearing station or a hospital near the front would differ very greatly from one written by a specialist attached to a base hospital, and still more from an account penned by an otologist at a military hospital at home. The first would deal mainly with cases of severe wounds of the head involving the ear, due to fragments from bursting shells or bombs or to rifle bullets—injuries which are fatal in a very high percentage of cases. On the other hand, otologists at home see comparatively few of these cases, but have to deal with indirect injuries of the ear caused by explosions or loud noises—i. e., with "shell" or "explosion" deafness, or "noise" deafness.

Wounds of the ear may lead to dangerous intracranial complications. Infected bullets or fragments of shell cause suppuration, especially when they carry in infected hair or skin. Secondary infection of the wounds is even more important than primary—i. e., bacteria can find their way to the injured middle ear from the nasopharynx via the eustachian tube or from the external meatus through a rupture of the drumhead.

The Middle Ear.—Rupture of the tympanic membrane is a fairly common war injury, and generally results from the explosion of a shell or bomb close to the sufferer. The lesion may be due to the sudden great increase of pressure which occurs first of all, or to the rapidly following negative pressure. The effect of the explosion varies with its distance from the ear, the size of the shell and the nature of the explosive. Lermoyez says that the effects of explosion are far greater in a trench than in the open field. The tears are often extensive and may give rise to the impression of actual loss of substance. The symptoms are slight bleeding from the ear, followed by serous discharge; there is also deafness and frequently vertigo and pulsating tinnitus. (The latter symptoms are, however, due to a concomitant lesion of the inner ear.) The question of infection is even more important than that of rupture of

the drumhead. Direct wounds of the middle ear always lead to extensive injury—usually to comminuted fractures of the petrous bone. It is only in rare cases that the middle ear alone is injured, and even then suppurative otitis media—usually of long duration—almost invariably follows. Frequently there is extensive splintering of the bone. The tympanic membrane and ossicles are torn or destroyed, and there is extensive hemorrhage into the drum cavity and antrum. The facial nerve is usually involved. In many cases the inner ear is also involved, and the lesion may extend to the intra-cranial structures. It rarely happens that shell fragments can remain in the mastoid without causing serious reaction.

The Labyrinth.—War injuries of the labyrinth may be classified as follows: (A) Direct injury due to bullets or fragments of shell or bomb. (B) Indirect injury in fractures of the skull. (C) "Noise" deafness, which is especially seen in artillerymen and naval gunners. (D) "Shell" or "explosion" deafness, due to the bursting of mines, shells or bombs close to the soldier.

(A) Direct Injury of the Labyrinth.—The symptoms of labyrinth injury are well known—deafness, tinnitus, dizziness. disturbance of balancing, vomiting, nystagmus, etc. Fifty per cent of the cases are killed outright at the time of the injury. Haymann states that in the other 50 per cent death occurs later from septic meningitis. The onset of meningitis may be delayed as long as six weeks.

(B) Indirect Injury of the Labyrinth in Fracture of the Skull.—The labyrinth may be indirectly injured by blows on the head from a rifle butt or by shot wounds of the head or face. The symptoms of such injuries are extreme or total deafness, marked disturbance of balancing, with loss of the cochlear and vestibular functions. If the labyrinth be injured there is an escape of cerebrospinal fluid from the ear along with blood. In the cases in which the labyrinth capsule is not injured there is no escape of cerebrospinal fluid; but, nevertheless, deafness results from hemorrhage into, or tearing of. the eighth nerve. On lumbar puncture blood may be present in the cerebrospinal fluid.

(C) "Noise" Deafness.—Wittmaack was the first to show that continuous noise conveyed to the ear by air or bone con-

duction did not cause hemorrhage or tearing of the membranous labyrinth but produced a degenerative neuritis in the cochlear apparatus. The vestibular apparatus showed no change. According to Wittmaack, the primary changes occurred in the spiral ganglion and nerve, while those in Corti's organ were only secondary. Yoshii, on repeating Wittmaack's experiments, found that Corti's organ was affected first of all, and that the degenerative changes in the cochlear ganglion and nerve followed later. Both von Eicken and Hoessli came to the conclusion that air conduction of sound was much more effective than bone conduction in producing injuries of the inner ear. After prolonged exposure to the sound of rifle and gun fire, deafness is usually marked and tinnitus not uncommon.

(D) "Shell" or "Explosion" Deafness.—The pathology of explosion or shell deafness is still very vague. (a) Some otologists believe that there are changes in the membranous labyrinth of a molecular nature leading to degenerative neuritis affecting the nerve endings, ganglia, etc. (b) Others hold that the membranous labyrinth is torn and that hemorrhages occur. Yoshii found that explosions produced close to the ears of animals caused rupture of the drumhead and hemorrhages in the cochlear and vestibular apparatus. Hoessli caused explosions at a greater distance from the animals and did not find rupture or hemorrhage but noted changes in Corti's organ, the cells of which were shrunken and flattened. (c) Zange has experimented on animals, but has found no changes in the labyrinth. He therefore believes that the condition is due to small hemorrhages in the pons, medulla and cerebellum.

<div align="center">WAR INJURIES.</div>

Case 1.—The case was one of shrapnel injury of the left ear followed by suppurative otitis media. When the patient was admitted to the Royal Infirmary, Edinburgh, about a month after the injury, symptoms of cerebellar abscess were present. Operation, performed by a general surgeon, revealed mastoiditis and small metallic particles in the mastoid antrum. In spite of the evacuation of the cerebellar abscess the patient died. The autopsy showed early meningitis and sinus thrombosis in addition to the cerebellar abscess. Microscopic exam-

ination of the ear demonstrated a thickened, infiltrated and
perforated drumhead (Figs. 31 and 32), with fracture of the
malleus. The lower part of the handle was separated from
the drumhead and drawn inward by the tensor tympani (Fig.
32). The mucosa of the middle ear was distinctly swollen
(Fig. 33), and the tympanic cavity, antrum and cells were
full of pus. The joint between the head of the malleus and
the body of the incus was dislocated (Fig. 30). The incudo-
stapedial joint was healthy, but there was a fracture of the
footplate of the stapes and early invasion of the vestibule
through the annular ligament (Fig. 32). The cochlea showed
hemorrhage in the scala tympani of the basal and middle coil
and in the opening of the perilymphatic aqueduct (Fig. 33),
but the nerve apparatus of the cochlea appeared almost
healthy. The neuroepithelium of the saccule and utricle and
of the cristæ of the canals was desquamating. The internal
meatus showed meningitis (Fig. 33).

Note.—Several of the changes noted in this case may have
been due to small pieces of shrapnel causing—e. g., fracture
of the malleus and stapes, hemorrhage in the scala tympani
and fracture of the bony capsule of ampulla of posterior canal
(Fig. 5).

Case 2.—Indirect injury of the ear, due to bullet wound of
the parietal ganglion. The patient was hit in the fronto-
parietal region, and the bullet came out just above the ear on
the same side. There was no apparent injury of the ear itself,
but the parietal bone was shattered and the brain lacerated.

Middle Ear Spaces.—Free blood is present in the lower part
of the tympanic cavity, in the niches of the round and oval
windows (Fig. 35) and in the mastoid antrum. Labyrinth
contents: The nervous structures of the inner ear appear to
be normal, but a little blood is present in the aqueduct of the
cochlea, near its cranial end, and also around the vestibular
nerve to the utricle. Internal meatus: There is a considera-
ble amount of hemorrhage within the arachnoid sheath which
surrounds the seventh and eighth nerve (Fig. 34).

Case 3.—High explosive shell injury. This soldier was
taking cover in a house when a high explosive shell
hit the house and burst into the room in which he was.

The patient was wounded in many places. On admission to the Casualty Clearing Station the patient was found to be semiconscious, with subnormal temperature, small rapid pulse, cold clammy skin and multiple small wounds. In fact, he showed the usual signs of high explosive shell injury, including marked deafness. This case shows certain definite changes—viz., rupture of the tympanic membrane (Fig. 36), slight hemorrhage in the middle ear spaces and in the fundus of the internal auditory meatus. The structures of the membranous labyrinth show normal conditions.

Case 4.—Injury to the ear due to the bursting of a rifle grenade. The grenade burst close to the left side of this soldier and caused extensive general injury. (Rifle grenades, though much smaller than shells, contain a very powerful form of high explosive.—J. Fraser.) The changes in this case of ear injury due to grenade explosion are fairly definite and include a recent rupture of the tympanic membrane, the edges of which are partially glued together by fibrin (Fig. 39). Further, there are hemorrhages in the tympanic cavity, in the marrow spaces in the roof of this cavity, around the geniculate ganglion and in the fossa subarcuata. There is also bleeding in the scala tympani of the cochlea in the region of the round window (Fig. 39). There is a marked hemorrhage in the fundus of the internal meatus. The otolith membranes of the saccule are separated (Figs. 37 and 38), and the neuroepithelium is irregular, while the cupula of the lateral canal is also separated (Fig. 40). Although the outline of Corti's organ is preserved, the hair cells and some of the supporting cells cannot be made out, so that the organ appears ghostlike.

Case 5. Shell Explosion.—The patient suffered from severe concussion, and the right side of the head and body presented multiple wounds. The changes in this case are very slight. This may possibly be explained by the presence of a plug of wax in the external meatus. There are small hemorrhages in the tubal part of the tympanic cavity, in the canal for the tensor tympani, in the cranial ends of the fossa subarcuata and perilymphatic aqueduct and in the fundus of the internal meatus. The neuroepithelium of the labyrinth shows little change, but the detachment of the otolith membrane of the

utricle and of the cupulæ of the lateral and superior canals is worth recording.

Case 6.—Injury by high explosive shell. This soldier caught the full blast of a high explosive shell, which burst close to his left side and shattered this side from head to foot. In this case the force of the explosion seems to have been expended in rupturing the drumhead (Fig. 41), so that the structures of the inner ear along with the nerves in the internal meatus have escaped. Owing to a fault in the first embedding of the specimen, air was present in the hollow spaces of the labyrinth, and it is therefore unwise to be dogmatic as to the condition of the membranous labyrinth. The rupture of the saccule and of portions of the membranous cochlea are probably artefacts produced by the use of the suction apparatus. The rupture to the posterior part of the annular ligament may also be due to the same cause.

Summary.—In four cases of "shell" or "explosion" deafness (Cases 3, 4, 5 and 6), the changes are as follows: (a) Rupture of the drumhead in Cases 3, 4 and 6. (In case 5 a large plug of wax was present in the external meatus, and probably for this reason the tympanic membrane was not injured.) (b) Hemorrhage in the middle ear spaces in all four cases. (c) Slight hemorrhage in the scala tympani in the region of the round window and basal coil of the cochlea (Case 4). (d) Slight degenerative neuritis of the cochlear apparatus—e. g., loss of hair cells, slight flattening of the acoustic papilla and disappearance of some of the supporting cells, especially in Case 4; shrinkage of the ganglion cells of the spiral ganglion; uneven staining of the cochlear nerve. (All these may be post-mortem changes.) (e) Displacement of the otolith membrane of the saccule and utricle (Cases 4 and 5), artefacts? (f) Displacement of the cupula of one or more of the canals (Cases 4 and 5). (g) The vestibular apparatus, as was to be expected, showed less change than the cochlear. The neuroepithelium of the saccule and utricle and the cells of the vestibular ganglion in the internal meatus appear to be better preserved than the corresponding parts of the cochlear apparatus. (h) Hemorrhage was observed in the fundus of the internal meatus at the points where the nerves enter the bony canals (Cases 3, 4 and 5).

5. LABYRINTHITIS.

Acute inflammatory affections of the labyrinth may be classified in various ways: thus, inflammation of the inner ear may be described as circumscribed or diffuse. Further, the diffuse form may be divided into stages. First of all, there is a serous, fibrinous or hemorrhagic stage. Later, the exudate in the labyrinth becomes purulent. The purulent stage is followed by the formation of granulation tissue in the hollow spaces of the inner ear—the granulations arising from the endosteum. Finally, the granulation tissue is organized into fibrinous connective tissue, in which more or less new bone formation occurs. Sometimes the process of ossification is so extensive that the whole of the hollow spaces of the inner ear are entirely obliterated.

Labyrinthitis may also be classified according to the causal disease or organism—e. g., labyrinthitis due to scarlatina, measles, influenza, etc. Further, labyrinthitis may arise either from acute or from chronic middle ear suppuration. In the former case the path of infection is almost invariably through the oval or round windows, whereas in the latter the labyrinth is usually invaded by the erosion of the prominence of the lateral semicircular canal by cholesteatoma formation. To put it in another way, in acute purulent otitis media the labyrinthitis is a diffuse one from its onset, whereas in chronic middle ear suppuration associated with cholesteatoma the labyrinthitis is first of all circumscribed and may remain so for a considerable period. An exacerbation of the middle ear suppuration, however, may lead to rapid spread of the circumscribed labyrinthitis, which thus becomes diffuse.

In order to illustrate the various types of simple inflammatory affection of the labyrinth it will be best to give a short account of the case history of the patients, followed by a description of the appearances found on microscopic examination of the inner ear after death.

CIRCUMSCRIBED LABYRINTHITIS.

Case 1.—George D., male, aged 4 years. Chronic middle ear suppuration (right) for three years, since scarlet fever. Pain in right ear, shivering, dizziness and vomiting for three days. Examination: Foul smelling discharge; red swelling behind right ear; mastoid tenderness; Rombergism marked. Tem-

perature 99. Tongue dry and furred. Scar of old gland abscess in neck. Watch heard on both mastoids. Conversation voice at two feet (R.), with finger closure of left ear. Nystagmus to side of lesion only. Operation on October 14, 1913. Mastoid cortex grayish white; large foul smelling abscess in mastoid, with extradural, perisinus abscess; dura yellowish gray and sloughy; sinus not opened; antrum full of cholesteatoma. Wound left open. Breathing stopped on one occasion during operation but restarted spontaneously. On the following day, temperature 101 at 8 p. m. Slight vomiting. Cerebrospinal fluid under pressure and contained polymorphs and organisms. Second operation on October 17th. Sinus further exposed, slit up and found thrombosed. Dark red clot; bleeding obtained from both ends. Jugular not ligatured; incision in cerebellar dura; gauze drain introduced. On following day Kernig's sign positive; child restless; early optic neuritis. Exploration of cerebellum without anesthetic; evacuated foul smelling cerebellar abscess. Urotropin given. On October. 27th, vomiting present; nystagmus to side of lesion. No reaction in wound. Coma supervened. Temperature 104. Death on October 31st. Postmortem: General purulent meningitis. Large cerebellar abscess. · Right internal jugular not thrombosed.

Microscopic examination of right ear: Round window niche largely filled up by connective tissue; cholesteatoma on inner wall of tympanum; granulations in roof of tympanum; caries of inner wall of attic. Erosion of bony wall of lateral canal (Fig. 42). Cochlea almost normal. A little pus in perilymphatic aqueduct. Purulent infiltration of modiolus. Utriculus and sacculus normal. Circumscribed labyrinthitis in lateral and posterior canals. In the latter there is new connective tissue formation with pus cells. The extradural abscess had evidently extended behind the labyrinth. Meningitis in internal meatus. Thrombus in jugular bulb.

Case 2.—Donald H., male, aged 23. Chronic middle ear suppuration (bilateral). Pain in right ear. Vomiting for two weeks, with shivering. Patient admitted to medical ward with diagnosis of "biliary colic." Rigor on day of admission (June 16, 1918). Temperature 103, pulse 116, respiration 36. Slight delirium.

Tongue dry and furred. Head retracted. Kernig present.
Nystagmus to both sides. Tenderness of right mastoid. Pa-
tient not completely deaf in right ear. Immediate operation.
Cerebrospinal fluid turbid and under tension (diplococcus).
Pus under pressure in antrum. Sinus healthy. Next day,
temperature 102. Rigor. Second operation: Right internal
jugular ligatured. Next day patient restless; severe head-
ache; heroin given. June 19th. Lumbar puncture. Cerebro-
spinal fluid under great tension and almost purulent; films
show many organisms. Fever continued, with rigors. Coma
supervened. Death June 23rd. (Patient had an uncle who
died from brain abscess.) Postmortem: Basal meningitis.
Temporosphenoidal abscess present, with thick walls. Abscess
had ruptured into lateral ventricle. Leakage into lateral ven-
tricle had probably occurred before admission. Sigmoid sinus
appeared healthy and therefore the rigors were probably due
to meningitis. (Blood cultures negative.)

Microscopic examination of right ear: Cholesteatoma in
tympanum, with thickened mucosa; new bone formation in
roof of aditus and antrum; bony wall of lateral canal eroded
(Fig. 43). Granulation tissue on inner wall and roof of an-
trum reached middle fossa—i. e., route of infection of brain
abscess; engorgement of marrow spaces in floor of middle
fossa, with absorption of bone; some marrow spaces contain
pus; cochlea normal except for a little pus at opening of peri-
lymphatic aqueduct. Slight cellular exudate in vestibule on
inner aspect of oval windows. Lateral canal shows circum-
scribed labyrinthitis. Meningitis in internal meatus.

CIRCUMSCRIBED FOLLOWED BY ACUTE PURULENT LABYRINTHITIS.

Case 3.—Annie B., female, aged 15 years. C. O. M. S.
(right). Noises in right ear and giddiness for one month; ear-
ache for one week; headache and pain in neck for five days;
vomiting for four days; shivering two days. Patient drowsy on
admission; slightly cyanosed. Temperature 98.6. Tongue furred
and dry. Right ear quite deaf with noise box in left ear.
Weber lateralized to better ear; slight spontaneous nystagmus
to left. Caloric reaction absent in right ear but fistula symp-
tom present. Cerebrospinal fluid clear but under increased
pressure. Kernig present; stiffness of neck; no optic neuritis.

Immediate operation: Foul perisinus abscess containing

gas; sinus wall sloughy; cholesteatoma in antrum; ossicles absent; fistula in lateral canal; sinus thrombosed; free bleeding obtained from torcular end; right internal jugular ligatured. At this point pulse very feeble. Labyrinth operation not performed. Saline injections given. During next week rigors continued and optic neuritis developed. Operation performed on jugular bulb. Death seventeen days after first operation. Postmortem: No reaction in wounds. Clot in right lateral sinus and also in superior longitudinal and straight sinuses. No meningitis or abscess of brain. Infarcts present in lungs.

Microscopic examination of right ear: There is rarefaction of the bone in the roof and floor of the tympanic cavity. Cochlear canal full of pus, whereas the scala vestibuli and scala tympani contain only fibrinopurulent exudate. Aqueduct of cochlea contains pus. The vestibule contains curdled lymph. Fistula present in lateral canal (Fig. 44); lumen of canal showed pus becoming organized. Other canals show purulent contents. Almost no meningitis in the internal meatus. The clot in the jugular bulb is becoming organized.

SEROUS LABYRINTHITIS.

Case 4.—John F., male, aged 11 years. C. O. M. S. (bilateral) for six years. Earache, headache and irritability for six days before admission. On examination, temperature 100; right mastoid tender; granulations and pus in right meatus; conversation voice at one foot (right ear); perforation of Shrapnell's membrane on left side; whisper at one foot (left); no nystagmus; cerebrospinal fluid clear and not under pressure; no photophobia. Three days after admission, temperature rose to 105°; head retracted; sudden facial paralysis on right side. Operation: Right mastoid contained cholesteatoma; sinus wall healthy; right temporosphenoidal lobe explored with negative result. Cerebrospinal fluid turbid on lumbar puncture; death two days after operation. Postmortem: Purulent meningitis at base; no brain abscess; venous sinuses healthy.

Microscopic examination of right ear: Cholesteatoma and polypus present in middle ear; bony exostosis from promontory; cochlea normal; vestibule healthy; slight serous labyrinthitis in lateral canal (Figs. 45 and 46); other canals normal. Meningitis in internal meatus, with slight hemorrhage and engorged vessels.

ACUTE SUPPURATIVE OTITIS MEDIA, PURULENT LABYRINTHITIS
AND LEPTOMENINGITIS WITHOUT RUPTURE OF THE
TYMPANIC MEMBRANE.

The following case shows that in a case of acute middle ear suppuration the pus in the tympanic cavity may penetrate into the labyrinth through the round and oval windows and from the labyrinth may infect the subarachnoid space before the purulent exudate in the tympanic cavity bursts through the tympanic membrane. John J., aged 12 years, was quite well till the morning of March 17, 1913, when he refused breakfast on account of severe earache and slight frontal headache. The boy vomited at 11 a. m. His doctor was called in and noted choreic movements of the hands. Vomiting continued at short intervals until the following morning. After this it was not so constant. The patient slept on and off during these first days of illness; he had frequent attacks of restlessness, and screamed if he were touched. At 1 p. m. on the day of admission (March 20th) he lapsed into a semicomatose condition.

Family History.—Father died of consumption at age of 36. One brother, aged 18, committed suicide three weeks before patient's illness began; this brother, who suffered from tubercular cervical adenitis, had been despondent for some time on account of severe deafness due to chronic suppurative otitis media.

Examination by Physician in Medical Ward (the boy was not seen by an otologist).—Temperature 102.4° F.; pulse 94. Conjugate deviation of eyes to the left. He moans constantly but there is no distinct cry. Kernig's sign present; no discharge from the ears; both tonsils large and red; abdomen slightly tense but not scaphoid. Lumbar puncture yielded milky fluid under considerable pressure. (Films showed many polymorphs and numerous capsulated diplococci, some in short chains; pneumococci on culture.) Next day Cheyne-Stokes respiration and death.

Postmortem.—Convolutions flattened; pus in subarachnoid space over both sides of cerebral cortex. Mastoid cells on both sides filled with pus. The left internal auditory meatus was also full of pus. On the right side the middle ear spaces contained pus. Ethmoid and sphenoid sinuses congested, but contained no pus. Hemorrhages in the pleura; bronchial

glands enlarged; on section right lung showed intense conges-
tion and small congested collapsed areas. The mucous mem-
brane of the trachea and large bronchi covered with blood
stained seropurulent fluid.

Microscopic Examination.—Right ear showed otitis media
but only hemorrhage in labyrinth (early stage of labyrinthitis)
(Fig. 47). Eustachian tube and tympanic cavity full of pus
(Fig. 50). The annular ligament is intact and is not infil-
trated with pus cells (Fig. 49). The secondary tympanic mem-
brane is moderately thickened but is not infiltrated (Fig. 51).
Cochlea: Hemorrhage in the scala tympani and scala media
in the neighborhood of the round window (Fig. 51). The
cochlear opening of the perilymphatic aqueduct contains pus
which has apparently found its way up the aqueduct from the
subarachnoid space (Fig. 51). Hemorrhage is present in the
osseous spiral lamina of the basal coil (Fig. 48). The modio-
lus and the nerve canals are infiltrated with pus, which appears
to have invaded from the subarachnoid space of the internal
meatus. There is a large hemorrhage in the perilymphatic
space of the vestibule (Fig: 49). Internal meatus: Pus is
present and surrounds the cochlear and vestibular nerves
(Fig. 47).

Left ear: The lining membrane of the eustachian tube is
swollen and the submucous tissue densely infiltrated with
small cells. Tympanic cavity filled with pus (Figs. 52 and
55); the mucous membrane over the promontory is greatly
thickened to form a long slender polypus. The posterior part
of the tympanic membrane bulges outwards towards the
meatus. The epidermic layer is desquamating and the fibrous
layer is disintegrated by small cell infiltration (Fig. 54). Pus
cells form a small abscess just beneath the desquamating epi-
dermic layer (myringitis). At this spot rupture of the mem-
brane was just about to occur. The footplate of the stapes is
normal, but the anterior part of the footplate is tilted outwards.
The annular ligament is infiltrated with pus cells, which are
finding their way through it into the vestibule (Fig. 53). All
coils of the cochlea contain pus (Fig. 52). The cochlear duct
contains pus, and Corti's organ is disintegrated. The modio-
lus is infiltrated with pus cells and the blood vessels are greatly
dilated. Round window: The secondary tympanic membrane

is greatly swollen and infiltrated with pus cells, which may be seen making their way through it into the scala tympani (Fig. 56). Vestibule: There is a considerable collection of pus just internal to the footplate of the stapes (Fig. 53).

ACUTE PURULENT LABYRINTHITIS; INFECTION THROUGH OVAL WINDOW.

Case 6.—Mrs. C., female, aged 20, gave a history of chronic suppurative otitis media (bilateral) since the age of 2 years. She had had pain in the right ear for six weeks before admission and vomiting for one week, with shivering. There was no dizziness. The patient was first seen on October 13, 1908, when it was noted that she looked ill and emaciated. The temperature was 102° F., and there was mastoid tenderness on the right side. The patient complained of pain in the occipital region and down the spine. The knee jerks were exaggerated. Next day facial paralysis (right) was present and third degree nystagmus to left; nystagmus also present on looking upwards. There was no ocular paralysis and the fundi were normal; knee jerks absent; grasp of right hand weaker than left.

Operation.—Mastoid cells and lateral sinus healthy; antrum contained granulations; dura internal to sinus showed granulations and softening; oval window empty; lateral canal healthy. Death occurred one day after operation. Postmortem: Basal meningitis.

Microscopic examination of right ear: Mucosa of middle ear greatly thickened and infiltrated, especially round windows (Fig. 57); fibrinous exudation in all scalæ, with few pus cells; basal coil most affected (Fig. 59); modiolus not infiltrated. Stapes displaced into vestibule (Fig. 57) and pus present here; oval window empty (Fig. 58). Empyema of saccus endolymphaticus. Purulent infiltration of canals. Meningitis in internal meatûs, with hemorrhage.

SUBACUTE LABYRINTHITIS LATENT.

Case 7.—Thomas G., male, aged 17 years, was admitted to a general surgical ward on September 5, 1911. He had suffered from chronic suppurative otitis media, right, since measles in childhood. Three days before admission the discharge suddenly ceased and pain in right ear commenced. On following

day swelling appeared behind the ear and the patient had a sudden attack of giddiness.

On examination (September 6th) : Abscess over mastoid ; temperature 100.4° F., pulse 100; sagging of posterior wall of the meatus.

The radical mastoid operation was performed. The patient was discharged from hospital one month after operation but was readmitted seventeen days later in a semicomatose condition. His face was pale; temperature 96° F.; pulse 68; pupils dilated and equal; they did not react to light. The patient lay curled up on the right side; incontinence of urine; almost stertorous breathing. The operation cavity was found to be full of pus. One hour after readmission the patient suddenly became flushed and perspired freely; pupils contracted; respiration gradually stopped but the heart continued to beat. Artificial respiration was carried out but the pulse became feeble and patient died. Postmortem: Abscess in right lobe of cerebellum.

Microscopic examination of right ear: Small celled infiltration of submucosa of middle-ear and new bone formation on promontory and in attic (Fig. 61) ; oval window not perforated ; polypus projecting from inner wall in region of lateral canal (Fig. 60) and hanging down into the tympanic cavity and external meatus. This polypus contains an abscess and communicated with a fistula in the lateral and posterior canals and also with an extradural abscess in the posterior fossa (Fig. 61). The polypus is covered with squamous epithelium except at the tip, where it is ulcerated. There is extensive softening of the labyrinth capsule, especially in the region of the canals (Fig. 61). The hollow spaces of the cochlea show curdled lymph and a little pus. There is also some new connective tissue formation in the scala tympani and scala vestibuli of all coils. The scala media is dilated and the spiral ligament atrophied. The vessels of the modiolus are distended. The spiral ganglion is atrophied and there is new connective tissue formation in the spiral canal. The saccule is dilated and contains pus and coagulum. The utricle is difficult to recognize. The perilymph space of the vestibule contains pus, which is becoming organized into granulation and connective tissue. There is even a little new bone formation next

to the endosteum. The canals contain pus, which is becoming organized into granulation tissue. The posterior canal shows slight new bone formation. The internal meatus shows small celled infiltration and also new formation of connective tissue. The vessels here are dilated. An extradural abscess is present in the region of the saccus endolymphaticus.

PANLABYRINTHITIS (I. E., PARA-, PERI- AND ENDO-LABYRINTHITIS).

Case 8.—Christina G., female, aged 6, had discharge from the left ear following an attack of measles at the age of 3 years. On December 25, 1911, she had severe pain in the left ear followed by vomiting, dizziness and headache (first attack of labyrinthitis). (This fact was not ascertained till after the death of the patient.) These symptoms gradually improved, and by January 8, 1912, she was fairly cheerful. When seen for the first time on January 13 the left external meatus contained pus and granulations; no mastoid tenderness. January 15, 1912. Adenoids and tonsils were removed. Unfortunately the granulations in the left ear were also curetted—a procedure which should be strictly avoided unless the labyrinthine function has been tested and found normal. January 17: Patient cyanosed and vomiting; temperature 103.2° F., pulse 160. January 18, complete deafness in left ear; dizziness present; rotatory and horizontal nystagmus to right (sound side); pulse feeble; tongue furred; diarrhea; cerebrospinal fluid under increased tension, but clear. (Films show streptococci and bacilli.) Operation considered hopeless. January 19, antistreptococcus serum given; facial paralysis present on left side; death.

Postmortem: Cloudy swelling of kidneys, heart and liver; petechial hemorrhages in lungs; enteritis; mesenteric glands showed recent acute inflammation in addition to chronic enlargement; no obvious meningitis. The appearances were those of acute septicemia.

Summary.—The case appears to have been one of septicemia and malignant serous meningitis following the removal of tonsils and adenoids and curettage of ear granulations in a patient suffering from latent labyrinth suppuration.

Examination of Left Ear.—Middle ear: Large perforation of the tympanic membrane (Fig. 65). Tympanum, aditus and

antrum lined by cholesteatoma (Figs. 62 and 65). There is caries of the bone beneath the cholesteatoma. On the inner wall of the attic there is a polypus just external to the ampullated end of the superior canal (Fig. 62). The malleus and incus have been displaced backwards (Figs. 64 and 65) (curetage). The head of the stapes is absent and the footplate necrotic and displaced. The annular ligament and the secondary tympanic membrane are swollen and infiltrated. Labyrinth capsule: There is extensive replacement of the cartilage bone of the labyrinth capsule by granulation tissue beginning in the inner wall of the attic. The disease has spread between the endosteal bone and the lamellar bone (Figs. 62 and 65). There are numerous osteoclasts in the granulation tissue. In the promontory the inflammatory process is invading the bone from the submucous tissue (Fig. 63). Cochlea: The basal coil shows delicate connective tissue and a little new bone in the scala tympani and vestibuli; there is great dilatation of the cochlear duct (Fig. 64). A trace of Corti's organ can be seen in the middle coil; elsewhere it is absent. The capsule of the cochlea shows several areas of "ostitis vasculosa," especially in the region of the anterior margin of the oval window (Fig. 63). The vestibule contains much pus (Figs. 63, 64 and 65). This is especially thick just internal to the oval window. The capsule of the vestibule looks as if it had been dissolved and replaced by granulation tissue. There is necrosis of the bony partition between the internal meatus and vestibule (Fig. 64). The membranous structures of the vestibule cannot be recognized. The canals show much the same changes. The granulation tissue around the posterior canal is in contact with the dura of the posterior cranial fossa. Internal meatus: Pus and granulation tissue are seen along the branches of the vestibular nerve (Figs. 63 and 64). The cochlear nerve is more normal. There appears to have been abscess formation in the posterior part of the meatal fundus, followed by the formation of granulation tissue, and probably by interference with the blood supply of the bony capsule of the vestibule and canals. This would favor the spread of ostitis (paralabyrinthitis).

From the clinical history it is evident that the patient had an attack of labyrinthitis on December 25, 1911. When seen for

the first time on January 13, 1912, the inner ear had passed into the stage of latent labyrinth suppuration. The paralabyrinthitis appears to have originated on the inner wall of the attic and was due to curettage of the aural polypus. The child probably died from septicemia due to infection following the operation on the fauces and pharynx in such a septic case.

SPONTANEOUS CURE OF LABYRINTHITIS.

Case 9.—Walter P., male, aged 42. · C. O. M. S. (left) since childhood. Loss of appetite and mental dullness for three weeks. Earache and dizziness for three days before admission to Workhouse Hospital. Headache on admission and tendency to fall backwards. Patient transferred, on account of irrational behavior, to the Royal Edinburgh Asylum on May 29, 1912. Tendon reflexes exaggerated on both sides, especially on left. General condition gradually improved. Wassermann reaction negative in blood and cerebrospinal fluid. On July 12, however, patient became confused and irritable. Death three days later. Postmortem: Acute endocarditis. Pachymeningitis externa over roof and posterior surface of left temporal bone. No brain abscess. The pathologist regarded endocarditis as probably secondary to the aural sepsis.

Microscopic examination of left ear: Extradural abscess above and behind labyrinth, affecting both middle and posterior fossæ (Fig. 67). Left drumhead retracted and adherent to inner wall of tympanum (Fig. 67). Drumhead shows small perforation in posterior part. Tympanic cavity almost obliterated. There is dense, small celled infiltration of submucosa over the promontory. Fistula present into basal coil of cochlea. There is some new bone formation in the wall of the tympanum. Head of malleus eroded; incus absent; stapes normal but embedded in thickened mucosa; oval window not perforated; niche of round window entirely filled up by connective tissue (Fig. 67). The hollow spaces of the labyrinth are almost entirely obliterated by connective tissue and new bone formation. There is pus present in the region of the crus commune, which communicated with the extradural abscess. This abscess extends over the convexity of the superior canal and has markedly eroded the bone in this region (Fig. 67). The extradural abscess also communicated by a band of fibrous tissue with the jugular bulb, which, however,

was not thrombosed. The middle and apical coils of the coch-
lea are less severely affected than the basal coil (Fig. 66),
which is entirely filled up by new bone. The scala media of
the middle coil is dilated and Corti's organ has disappeared.
The scala tympani and scala vestibuli show some new fibrous
tissue and bone formation. Further, the opening of the peri-
lymphatic aqueduct is filled up by new bone. The nerve canals
of the modiolus are filled up with new fibrous tissue; nerves
and ganglia absent. Vestibule largely filled up by fibrous tis-
sue (Fig. 67). Utricle, saccule and endolymphatic aqueduct
not recognizable. The canals are less affected than other
parts, but the perilymphatic space contains pus and some new
formed fibrous tissue. Dura of internal meatus greatly thick-
ened (Fig. 66). Abscess present in floor of meatus. The ex-
tradural abscess is in contact with the dura of the roof of the
meatus. The seventh and eighth nerves are incorporated in
fibrous tissue. Only traces of the vestibular ganglion can be
seen.

Case 10.—William N., male, aged 26 years, had suffered from
chronic suppurative otitis media (left) since childhood. He was
admitted to a general surgical ward with symptoms of cerebellar
abscess. The cerebellum was explored behind the sigmoid
sinus, with negative result. Ten days later the patient had a
rigor, and at the second operation the sigmoid sinus was found
to be thrombosed. The jugular vein was ligatured but the
radical mastoid operation was not performed. The patient
made a temporary recovery. Later he was readmitted on ac-
count of ear discharge and sent over to the Ear and Throat
department. Examination on admission showed slow pulse
and subnormal temperature. The patient was drowsy but did
not vomit and had no headache; no tenderness over skull; no
sensory aphasia; no paralysis; marked Rombergism; complete
deafness on the left side; spontaneous nystagmus to right
(sound) side; caloric reaction absent on left side but prompt
response obtained on right side.

Third Operation (radical mastoid).—Cholesteatoma in mid-
dle ear spaces; lateral canal showed no fistula but had lost the
usual white appearance. Labyrinth operation not performed.
Eleven days later nystagmus still present but no vomiting.
On fifteenth day after the third operation headache and vomit-

ing were present; optic discs normal; cerebrospinal fluid clear. The patient died suddenly next day. Postmortem: Cerebellum adherent to dura in triangular area; abscess in left lobe of cerebellum, with surrounding edema; no meningitis.

Microscopic examination of left ear: Cholesteatoma in attic and aditus; stapes displaced into vestibule (Fig. 69)—probably by contraction of fibrous tissue; round window membrane thickened (Fig. 69); labyrinth capsule very vascular. The hollow spaces of the inner ear are filled up by connective tissue and new bone (Fig. 68). There is, however, still some pus (abscess) in the center of the vestibule (Fig. 70). The membranous labyrinth has disappeared. The internal meatus shows the remains of the seventh and eighth nerves surrounded by vascular fibrous tissue and old hemorrhage (Fig. 68). The jugular bulb contains organized clot.

Case 11.—William A., male, aged 5 years, had had discharge from right ear for more than one year; a large mastoid swelling for two days. Examination showed sagging of the roof of the meatus on the right side. The right ear was quite deaf when the noise apparatus was going in the left. There was no spontaneous nystagmus. Cold syringing of the right ear produced no reaction. Temperature 100.6°; opacity of right cornea. The patient had been attending a tuberculous hospital.

Operation.—Pus in antrum; roughness of prominence of lateral semicircular canal but no fistula. On curetting the promontory the small spoon passed into the vestibule, which was therefore freely opened up. Dura of posterior fossa exposed and attempt made to open up posterior and lateral canals; these, however, could not be found. The facial nerve twitched on two occasions. On the following day facial paralysis was noted; Kernig's sign present; temperature 103° F.; nystagmus to side of operation; photophobia. Sedatives were given. Later there was marked retraction of the head and opisthotonus; scaphoid abdomen. Death occurred four days after operation. Postmortem: Caseous bronchial glands; large hemorrhage in left parietal region (side opposite to operation); basal meningitis; left mastoid healthy.

Microscopic examination of right ear: Complete filling up of hollow spaces of labyrinth by new bone formation (Fig. 71). There is evidence of bony softening in region of ves-

tibule and basal coil of cochlea, in the spot where the spoon entered at the operation.

Case 12.—Margaret W., female, aged 52. Patient gave a history of chronic suppurative otitis media (right) for over twenty years. Two weeks before admission she had pain in the right ear and four days later a rigor and vomiting. The patient's doctor suspected typhoid.

On examination, no headache or mastoid tenderness; temperature 105° F. Operation: Pus and cholesteatoma in antrum; sigmoid sinus brownish black; fetid pus escaped; free bleeding obtained from torcular end. Internal jugular vein exposed and found to be collapsed; apparently healthy clot reached just above the clavicle. Progress: No reaction in wound; rigors recurred, with vomiting. Blood culture showed streptococcus and antistreptococcus serum was given. Death occurred fourteen days after operation. Postmortem: Septic thrombus in lower end of internal jugular vein; infarcts and abscesses in lung.

Microscopic examination of right ear: Mucosa of middle ear thickened and engorged; cholesteatoma present; some new bone formation on promontory; annular ligament intact but stapes footplate eroded (Fig. 73); bone around labyrinth capsule very vascular; granulation tissue present between posterior canal and posterior fossa (extradural abscess); cochlea entirely replaced by bone (Fig. 72); round window cannot be recognized though the niche can be seen; vestibule shows great dilatation of saccule and utricle (Fig. 73); neuroepithelium of utricle present; new connective tissue and a little bone formation in perilymph space of vestibule. There is evidence of an old fistula in the lateral canal; connective tissue present in crus commune and in other parts of the canals. Internal meatus shows small hemorrhage along the vestibular nerve; cochlear nerve completely atrophied (Fig. 72); facial nerve normal.

6. TUBERCULOSIS OF THE MIDDLE EAR AND LABYRINTH.

Note.—The following remarks on the clinical aspect of tuberculous disease of the ear have been abstracted from an article published by Dr. Logan Turner, Journal of Laryngology, Rhinology and Otology, Vol. XXX, 1915, page 209:

Age Incidence.—Of 60 cases, 51 were children and 9 adults.

During the period 1907-14 the cases of middle ear suppuration under fifteen years of age, seen at the Ear and Throat Department of the Royal Infirmary, Edinburgh, numbered 1,797, of which 51, or 2 per cent, were tuberculous. Among these 1,797 cases there were 505 cases of middle ear suppuration under five years and, of these, 48 were tuberculous—i. e., 9 per cent. Under the age of two years there were 172 cases of middle ear suppuration, of which 47 were tubercular—27 per cent. In the first year of life there were only 86 cases of middle ear suppuration, and 43—i. e., 50 per cent—were tuberculous. In 32 of the 43 infants there was evidence of the disease in the first six months of life.

Etiology (in infants and young children).—The majority of the children suffering from tuberculosis of the ear were "bottle fed," and it was most exceptional to obtain any history from the mother of the milk having been boiled or sterilized. With regard to breast fed babies it was a common experience to hear that the breast feeding was supplemented by bottle milk. Mitchell has examined 406 samples of mixed milk collected from the same number of milk shops in Edinburgh; in 82 samples, or 20 per cent, tubercle bacilli were found. (In every instance the inoculation of guinea pigs was the method employed for the detection of tubercle bacilli.) Further, 72 consecutive cases of tuberculous cervical glands in children were investigated by Mitchell. The bovine type of the tubercle bacillus was present in 65 instances (90 per cent) and the human bacillus in only 7 (10 per cent). Eighty-four per cent of the children had been fed on unsterilized cow's milk since birth. John Fraser has examined 70 cases of tuberculous bone disease and found in 60 per cent of the cases the bovine strain of tubercle bacillus.

The second type of tubercular otitis media occurs in the advanced stages of phthisis pulmonalis. St. Clair Thomson found only two cases among 700 patients suffering from pulmonary tuberculosis. Herzog found chronic purulent otitis media in 17 out of 100 cases of "consumption"; 5 of the 17 cases showed labyrinthine involvement. Turner and Fraser have examined 5 cases of purulent otitis media occurring among 120 cases of phthisis pulmonalis (mostly of the ad-

vanced stage). In only one of these five cases was there any suspicion of middle ear tuberculosis.

The Path of Infection.—In the majority of cases of middle ear tuberculosis in children the infection reaches the ear through the eustachian tube. This may occur either by the infectious material passing through the lumen of the tube without involving its lining membrane or by the direct extension of the tuberculous disease from the nasopharynx along the lining membrane of the eustachian tube. There may be evidence of the existence of tuberculous disease in the adenoid vegetations of children affected with tubercle of the middle ear cleft. Infection of the temporal bone by the blood stream is probably rare in children. Henrici and others, however, hold that tubercular otitis is often due to a hematogenous infection. If it can be shown at operation that the tube and tympanum are healthy, and that the mastoid alone is diseased, the probability is that the infection has occurred by way of the blood stream.

Clinical Characteristics of Middle Ear Tuberculosis. (a) Mode of Origin.—This is, as a rule, of a quiescent character, and in 92 per cent no history of apparent pain or restlessness could be obtained either preceding or accompanying the onset of the ear affection. The mother's attention is first drawn to the ear by observing the presence of discharge. Enlarged periotic glands were noted in 95 per cent of the cases. In a number of these caseation and suppuration occurred.

The discharge from the ear is watery or flocculent—at least in the early stages before mixed infection occurs. Multiple perforations in the drumhead may be observed in adults, but in infants the sagging of the meatal will usually prevents inspection of the tympanic membrane. If mixed infection occurs the symptoms of an acute otitis media and mastoiditis may arise. Facial paralysis was present in 45 per cent of the cases observed by Turner.

Types (or Stages) of Tuberculous Otitis Media and Interna.—(1) Lupoid—in cases of lupus of the nose and throat. (2) An infiltrating form, which progresses rapidly and shows numerous tubercle bacilli but few giant cells. (3) A fungating or more chronic form with well marked tubercle follicles and few bacilli. (4) A necrotic form which tends to rapid caseation and destruction of the mucosa and bony capsule of

the labyrinth. The necrosis may be due to blocking of one or
more of the arterial twigs supplying the labyrinth capsule by
tuberculous endo- or peri-arteritis, but mixed infection is prob-
ably of even greater importance. (5) A fibro-ossifying type of
tuberculous otitis interna may occur—i. e., a type in which
nature makes considerable effort towards a spontaneous cure
of tuberculous labyrinthitis by the formation of new fibrous
tissue and bone. This type of tubercular disease is well recog-
nized in the case of the long bones.

Pathologic Conditions Found at Operation.—Carious bone
was found in 65 per cent and a sequestrum in 60 per cent.
In some cases the outer antral wall lay loose and could be read-
ily picked out with forceps. The granulations often presented
a pale flabby appearance. Caseous material was noted in the
antrum and tympanum in several cases. In nine instances the
malleus and incus were absent.

Concomitant Affection of the Labyrinth.—In 35 of the
series the mastoid operation was performed and the labyrinth
was found to be necrosed in whole or in part in eight of these,
or in 22 per cent. In addition, the external labyrinthine wall
showed pathologic changes in 11 cases—i. e., 19 out of 35
cases showed labyrinthine changes (54 per cent). The lesions
observed were superficial erosion and softening in the region
of the external semicircular canal, facial canal and promontory.

In infants and very young children the ordinary tests of the
labyrinth function can hardly be applied, but if the radical
mastoid operation be performed it is possible, at the end of the
operation, to apply cold lotion to the inner wall of the oper-
ation cavity, and the anesthetist is able to observe whether or
not this procedure results in conjugate deviation of the eyes
to the lower or nonoperated side.

Route of Infection from Middle Ear to Labyrinth.—Infec-
tion spreads to the labyrinth from the middle ear, as a rule,
by way of the oval or round windows. The stapes footplate
is soon eroded, but the endosteum lining the vestibule resists,
and is pushed inwards in front of the mass of tuberculous
granulation tissue before rupture occurs and the perilymph
space of the vestibule is invaded. Invasion of the labyrinth
through the secondary tympanic membrane is not resisted to
the same extent. The prominence of the external semicircular

canal is occasionally the seat of a fistula into the labyrinth, but not nearly so often as in cases of cholesteatoma. On the other hand, the promontory is much more often diseased in tubercular than in simple purulent otitis media. Clinically, tubercular labyrinthitis, like tuberculous otitis media, appears to have a quiet onset.

Complications: Tuberculous pachymeningitis externa is frequently met with at operation. Hemorrhage from the internal carotid artery has been recorded. Tubercular thrombosis of the sigmoid sinus may occur. Tuberculous meningitis and tuberculous tumors are met with at postmortem examinations but they are usually regarded as parts of a general tuberculosis. Two of the patients who were operated upon died of tuberculous meningitis.

Treatment.—Brieger holds that the indications for treatment in tuberculosis of the middle ear are the same as those in the ordinary purulent forms of otitis media and mastoiditis. Ruttin has informed us that in Vienna they only operate on tuberculous otitis if the patient is suffering severe pain, as they consider it hopeless to get rid of all the disease. Probably the correct position lies between these two extremes. It is useless to perform a Schwartze operation. The radical operation is therefore indicated in all cases except those rare forms in which there is an apparently hemotagenous involvement of the mastoid process. At the radical operation it is advisable to expose freely the dura of the middle and posterior fossæ, because this membrane forms a much better barrier than the bone to the spread of tubercular disease. One can curette the eustachian tube from the mastoid wound, and remove tubercular adenoids if they be present. It is, therefore, not possible to get rid of the whole of the disease, so that one must depend to a great extent on the resisting powers of the patient, and for this reason it is very important that sanatoria should be provided for the after treatment of the surgical forms of tuberculosis. It is useless to send these cases of tuberculous otitis back to the slums from which so many of them come.

Pfannenstiel's method of treating tuberculosis of the nasal mucous membrane may be applied with advantage in the after treatment of tubercular otitis media.

Case 1.—W. Y., aged one year and three months; bottle fed; bilateral tuberculous otitis media; left facial paralysis one month before admission. Child has never had any pain in the ear. Foul pus in both meatus; shotty glands behind sterno-mastoid.

Operation on Left Ear: Antrum contained granulation tissue; bone around soft, and granulations in oval and round windows; malleus and incus absent. Later, operation on right ear. Two days after this second operation the child became very restless; rapid respiration; squinting; vomiting; broncho-pneumonia present. Death.

Postmortem.—Tuberculosis of right lung and of bronchial glands; mesenteric glands enlarged and caseous; no tubercle of brain. (Guinea pigs inoculated from lymphatic glands removed from back of left ear showed definite tuberculosis. Granulation tissue from the ears showed small tubercular areas.)

Microscopic Examination of Right Ear (Case 1).—Eustachian tube: Tuberculous nodules present in submucosa. Tympanic cavity: Tuberculous granulation tissue is seen extending into the roof of the tympanum (Fig. 74) and the inner wall of the antrum, and can be traced from this for some distance through the nucleus of the labyrinth (fossa subarcuata). Oval window: The stapes is eroded and displaced towards the vestibule (Fig. 75). There is granulation tissue present between the footplate of the stapes and the endosteum of the vestibule. At the lower margin of the oval window the annular ligament is disintegrated, the endosteum is ruptured and the purulent infiltration has entered the perilymph space of the vestibule, and, penetrating Reissner's membrane, has entered the cochlear canal. Round window: The secondary tympanic membrane is greatly thickened, the niche of the round window being filled with swollen mucosa containing tubercular granulation tissue (Fig. 75). Labyrinth capsule: The lamellar bone over the promontory is eroded, but the cartilage bone is almost entirely healthy. The bony wall of the external canal prominence is also eroded (Fig. 76), but a fistula has not formed. Cochlea: There are curdled lymph and mononuclear cells in the scala tympani of the basal coil, especially just above

the round window membrane (Fig. 75). Vestibule: Just above and internal to the oval window the branch of the vestibular nerve to the utricle and cristæ of the external superior canals is infiltrated with pus cells.

Remarks.—The case was an early one of infiltration tuberculosis affecting the mucosa of the middle ear cleft. The infection had apparently spread up the tube and involved the mucosa of the tympanum, attic, and mastoid antrum; from the inner wall of the antrum it had spread along the vessels of the fossa subarcuata towards the cranium. Invasion of the labyrinth was just beginning.

Case 2.—J. N., male, aged nine months (a twin), had epileptic fits at the age of six weeks; a fortnight later the right ear began to discharge; the fits continued at intervals. At three months the right ear was operated upon at another hospital, but the discharge continued. Examination.—Child markedly wasted; mouth shows thrush; frequent vomiting. Child's head sweats profusely and is markedly retracted; photophobia present. Enlarged glands below the right ear, and behind the ear there is a fistula discharging pus. The right side of the face is paralyzed.

Operation: Large flat sequestrum removed from roof of middle ear. Sequestrum also removed from floor and another from the inner wall, opening up the cochlea, vestibule, semicircular canals and fallopian aqueduct.

After operation progressive emaciation and glandular enlargement; wound cavity shows few granulations. Death two weeks after operation.

Postmortem: Tuberclular nodule in right anterior corpus quadrigeminum and another in the occipital region. The dura of the right middle fossa showed tubercular thickening.

Microscopic Examination of Right Ear (Case 2).—Eustachian tube: The walls of the bony portion of the eustachian tube are destroyed, so that the dura (above) and the internal carotid artery (to the inner side and below) are infiltrated with tubercular tissue (Figs. 77 and 78). Tympanic cavity: The anterior part of the tympanum shows marked tubercular infiltration with caseation. The bony wall (cochlear capsule) is eroded—both lamellar and cartilage bone being involved

(Fig. 79). Oval and round windows: Absent through dis-
ease. The marrow spaces show tubercular granulation tissue,
which extends up to the dura mater of the posterior fossa.
Cochlea: All coils are filled with granulation tissue. No giant
cell systems are seen, but there is a good deal of caseation.
All membranous structures have disappeared. The outer wall
of the cochlea is almost entirely eaten away. There is a fistula
from the middle ear into all three scalæ of the cochlea (Fig.
79). (From the appearances present it is evident that there
was a mixed infection of the labyrinth in this case.) Vestibule
and Canals: There is a large fistula from the middle ear into
the vestibule, and all membranous structures have disappeared
except the aqueduct which, at the vestibular end, is plugged
with granulation tissue and pus. The nucleus of the labyrinth
has broken down into a tuberculous abscess, and the disease
has spread along the fossa subarcuata (Fig. 80). The external
canal has disappeared entirely, while the posterior canal con-
tains pus in both ends. Internal Meatus: Shows greatly
engorged and dilated vessels; there are a few pus cells in the
fundus of the internal meatus.

Remarks.—This case was one of the severe type of tuber-
cular otitis media and interna—the necrotic form. In spite
of the large area of necrosis, which included the promontory,
the external canal, part of the superior and posterior canals and
almost the whole of the fallopian aqueduct, there is no evi-
dence of gross disease of the labyrinthine artery in the inter-
nal meatus. The extensive necrosis seems to have been due
to the mixed infection rather than to any gross disease of the
artery supplying the labyrinth capsule.

Case 3.—H. C., male, aged nine months (breast fed). Nasal
catarrh and obstruction since birth; left sided otorrhea without
pain of ten weeks' duration; swelling behind left ear for eight
weeks. Wilde's incision made by patient's doctor. Examina-
tion: Very marked adenoids; large kidney shaped perforation
of left tympanic membrane.

Adenoid operation performed and later the radical mastoid
operation. Much granulation tissue present. Head retraction
developed; meningitic cry; internal strabismus; cerebrospinal
fluid clear—slight increase in mononuclear cells. Death.

Postmortem: Tubercular meningitis and general tuberculosis.

Microscopic Examination of Left Ear (Case 3). Middle Ear.—The mucous membrane is greatly thickened and infiltrated with tubercle over the lower part of the promontory and in the niche of the round window. In the latter position giant cell systems are present. The stapes is absent and there is a large fistula into the labyrinth through the oval window (Fig. 83). There is a second fistula between the scala vestibuli of the basal coil of the cochlea and the tympanic cavity (Fig. 81). The round window membrane is greatly thickened and infiltrated. Cochlea: The basal coil of the cochlea in its lower part is entirely filled up with new connective tissue and bone; the new bone formation is especially marked in the scala tympani and occludes the opening of the perilymphatic duct. There is some new endosteal bone in the scala vestibuli. The spiral canal in the basal coil is filled up with small cells and the ganglion cells have disappeared. No trace of Corti's organ remains. In the middle coil there is formation of new connective tissue with a little new bone in the scala tympani. The cochlear canal itself is greatly dilated by contraction of the new connective tissue in the scala vestibuli, pulling on Reissner's membrane (Fig. 82). In the middle coil Corti's organ is represented by a low mound of cells in which pillar cells cannot be recognized. The modiolus is markedly infiltrated with small cells. Vestibule and canals: The vestibule shows a considerable amount of new fibrous tissue and bone formation from the endosteum (Figs. 83 and 84). There is a large cavity in the center, lined by tubercular granulation tissue which shows giant cells in parts and in others caseation and sequestrum formation. No membranous structures are visible. The canals are also entirely replaced by new formation of connective tissue and bone. Internal meatus: The nerves are infiltrated by small mononuclear cells. In parts the infiltration amounts to nodule formation (Fig. 81).

Remarks.—The well marked tubercle follicles present in the tympanic mucosa in this case point to a chronic form of tuberculosis. The condition of the labyrinth is interesting, as there is great evidence of an attempt on the part of nature to achieve a spontaneous cure of tubercular labyrinthitis by a process of

fibrosis and new bone formation. The invasion of the laby-
rinth appears to have taken place mainly through the oval
window into the vestibule and, to a less extent, through the
round window into the cochlea.

7. NEURITIS OF THE EIGHTH NERVE (MÉNIÈRE'S SYNDROME).

Ménière, in 1861, described the case of a young girl who,
after a cold drive during her menstrual period, was attacked
by sudden deafness, intense dizziness and vomiting. She died
on the fifth day of her illness, and at the autopsy it was found
that the semicircular canals and the vestibule contained hemor-
rhagic exudate. The cause of death was not discovered.

Although Ménière's classical case was described more than
sixty years ago, there is still considerable confusion on the
subject of Ménière's disease, and the term is frequently ap-
plied to any condition in which dizziness is associated with a
lesion of the ear. It would probably be better to discard alto-
gether the use of the words "Ménière's disease," but, if the
term be retained, it should be kept for cases in which Ménière's
symptoms appear in an apoplectiform manner.

These symptoms include "deafness, tinnitus, giddiness, nau-
sea, vomiting, loss of balancing and nystagmus." This "dis-
ease picture" is that of a lesion of the membranous labyrinth
or eighth cranial nerve. It should be associated with disease
of the labyrinth, just as "tremor, rapid pulse and exophthal-
mos" are associated with the condition of hyperthyroidism.

Apart from injuries of the labyrinth—e. g., fracture of the
cranial base, war injuries, etc.—most of the cases of Ménière's
symptom complex are probably due to neuritis of the eighth
nerve, associated with toxemia or exposure to cold. Except
in the bleeding diseases—e. g., leukemia and pernicious ane-
mia, etc.—hemorrhage into the labyrinth has never been
proved by microscopic examination.

As syphilis is a frequent cause of Ménière's syndrome, the
Wassermann reaction of the blood and cerebrospinal fluid
should always be tested. Tabetic auditory nerve deafness is
one of the less common complications of locomotor ataxia and
may be due to a degenerative neuritis in the nerve trunk or
an affection of the auditory nucleus. Lehmann has found that
nerve deafness of toxic origin may occur in enteric fever; the
vestibular apparatus is not affected, while permanent injury to

the cochlear nerve is rare. Recently recurring attacks of Ménière's symptoms have been attributed to toxic neuritis or neurolabyrinthitis due to absorption of toxin from a focus of infection—e. g., pyorrhea alveolaris, septic tonsils, appendicitis or to gastrointestinal disturbances in people whose auditory or vestibular apparatus is specially sensitive. Ruttin has pointed out that as the lymphatic spaces of the inner ear are continuous it is almost impossible to conceive of an isolated affection of the cochlear or vestibular apparatus arising within the labyrinth itself. On the other hand, the cochlear and vestibular nerves may be affected separately by toxins which have a special affinity for one or the other division. When the cochlear apparatus alone is affected we have tinnitus and nerve deafness. When the vestibular apparatus alone is involved dizziness is the chief symptom. The complete Ménière syndrome is present only when the labyrinth or both parts of the eighth nerve are affected. In chronic nephritis labyrinthine deafness is due to a toxic rather than an inflammatory, circulatory or edematous change. It is usually associated with a very considerable amount of · vertigo. Degenerative neuritis occurs in cases of diabetes mellitus—the cells of the spiral ganglion being first affected. A similar neuritis has been described in gout and in cancerous cachexia. Toxic paralysis may be caused by quinin, salicin or salicylates, more rarely by alcohol, tobacco or lead poisoning. In many instances, however, the etiology of the neuritis is obscure.

A Case Presenting Ménière's Symptoms Along with Facial Paralysis.—A. McR., male, piermaster, aged 65, had never suffered from deafness up to the time of his last illness. On January 12, 1907, he had a long day of exposure to cold on the pier. The following morning he woke up suffering from extreme dizziness, deafness and noises in the left ear. The left side of his face was paralyzed. He also complained of pain in the head and limbs and did not leave his bed for a week. He felt as if surrounding objects were moving around him. There was no herpes auricularis. When he got up he had to hold on to furniture in order to get about the house. Examination November 2, 1907: Tympanic membrane normal, watch heard by right ear at two inches; by the left it is not heard even on contact. Forced whisper heard by right ear at

two feet and by left ear at one foot. All tuning forks heard by air conduction by left ear. Rinne's test positive on both sides. Patient can stand with his feet together and eyes shut without swaying. Left facial paralysis present, but patient can taste salt on the anterior part of the tongue on the left side. No response to faradic current on the left side of the face; the affected muscles contract slowly to galvanism, but there is no alteration of the polar reaction. The arterial walls are thickened. The lungs both reveal extensive chronic tuberculosis. Joints of hands swollen and painful. The diagnosis made by the late Dr. Alexander Bruce was that of neuritis of facial and auditory nerves in the internal auditory meatus. "The absence of evidence of middle ear disease and the retention of the sense of taste, and the associated deafness, dizziness and facial paralysis indicated that the lesion was probably situated between the side of the pons and the bottom of the internal auditory meatus."

The patient died suddenly February 5, 1908.

Postmortem: Atrophy of second, third, fourth and sixth cranial nerves on left side; extensive chronic pulmonary tuberculosis; subacute interstitial nephritis; milky thickening of arachnoid over inferior surface of cerebellum and circle of Willis.

Examination of left inner ear and auditory and facial nerves: The inner ear was almost normal; there was no hemorrhage in the endo- or peri-lymphatic spaces of the cochlea, vestibule or canals. The membranous labyrinth was also normal, as was the spiral ganglion. The neuro-epithelium of the utricle, saccule and of the cristæ ampullarum was well preserved. In the internal meatus there was a considerable amount of small cell infiltration (Fig. 85), between the epineurium and the seventh and eighth nerves; this was most marked above the seventh nerve. The vessels in the internal auditory meatus were markedly thickened, and the muscular coat had undergone a hyalin change. The infiltration of white cells did not extend into the Fallopian canal and did not therefore affect the geniculate ganglion.

8. TUMORS OF THE EIGHTH NERVE.

In the vast majority of if not in all cases the growth starts in the internal auditory meatus and only later invades the

cranial cavity. Cushing points out that owing to the characteristic chronology of their symptoms, acoustic tumors may, as a rule, be sharply distinguished from all others. The writer hopes that in the future otologists may be able to diagnose these cases in the early or otologic stage, when operative treatment holds out a much better chance of recovery than exists at the later periods when, according to Cushing, all we can effect is an extensive decompression operation combined with removal of "as much as possible" of the growth.

Etiology: (1) Heredity.—Neurofibroma of the eighth nerve is related to general neurofibromatosis, which may occur as a family disease. Feiling and Ward have published a paper on a "familial form of acoustic tumor."

(2) Age.—Henschen has collected 125 cases; second decade, 9; third, 20; fourth, 26; fifth, 27; sixth, 6; seventh, 1. The symptoms of an acoustic tumor rarely occur before the third decade.

(3) Infection.—Hessler has collected nineteen examples of acoustic tumor in patients who had suffered from middle ear disease. Siebenmann and Nager, on the other hand, believe that these acoustic tumors are associated with intrauterine meningitis.

Pathology.—Virchow has classified neuromata into (1) true and (2) false. Some say that true neuromata do not exist. False neuromata are composed of connective tissue derived from the nerve sheath and include acoustic neurofibromata. The endoneural connective tissue between the fibers is the seat of the pathologic process. The perineurium at first remains unchanged and forms the capsule of the tumor. Henschen states that five cases of "solitary" but symmetrical tumors of both eighth nerves have been recorded, and further, that the cases of multiple neurofibromatosis with acoustic tumor number sixteen.

The tumors vary in size from a hazelnut to a small apple. They have an uneven nodular surface and a grayish pink color. On section the tumors may show cysts or spaces filled with blood.

Histology.—Of 107 cases collected by Henschen, 86 showed a connective tissue structure and 21 appeared to be gliomatous. The latter appearance may be due to edema. Greenfield

states that the tumor is composed of cells of the connective tissue type. These cells have an elongated nucleus and the cytoplasm tails off into a spindle shaped process at either end. There are areas of looser texture, suggesting myxomatous or glial tissue. They are probably mixed tumors, glio-fibromas composed of germinal tissue—i. e., they are tissue "rests." They probably spring from the crest of nerve tissue lying between the ectoderm and the dorsal surface of the neural tube from which the seventh, eighth, ninth and tenth nerves arise. Henschen believes that the tumors originate in the inferior vestibular branch of the distal portion of the nerve in the internal meatus. He points out that between the branches of the eighth nerve in the internal meatus there is present embryonic connective tissue and that this explains why the eighth nerve is so often the seat of tumor. Orzechowski, on the other hand, thinks that the tumors are gliomatous and have little or nothing to do with the acoustic nerve and holds that they proceed from a malformation of the median wall of the lateral recess. Bruce and Dawson came to the conclusion that fusiform nucleated cells, linked on to one another, form embryonic nerve fibers—i. e., that nerve fibers are formed by the same process which gives rise to neuromata in the repair of divided nerves. A neuroma thus arises from the perverted function of the neurilemma sheath.

The eighth nerve consists of two parts: (a) A central non-medullated, and (b) a peripheral medullated portion. The junction of these two parts occurs just within the internal auditory meatus at its cranial end. Henschen states that in all cases of tumor only the distal portion of the nerve was affected, just beyond the junction of the two parts. In the case of large tumors there is always a cone shaped portion of the growth in the internal meatus.

Changes in the Labyrinth.—Alexander and Fränkl-Hochwart have examined the labyrinth in one case of acoustic neuroma and found degenerative atrophy of the cochlear nerve and spiral ganglion; atrophy of Corti's organ and the stria vascularis; dropsical degeneration of the spiral ligament. Alexander in another case found the round window membrane thickened by a layer of connective tissue on its cochlear side. The perilymph spaces contained a network of fibrin threads (Alex-

ander thinks that this fibrin network becomes organized into connective tissue) ; the bony spiral lamina filled with connective tissue; all the labyrinth nerve endings were degenerated. Corti's organ was absent in parts. Frankenstein in a case of multiple neurofibromatosis found the hollow spaces of the modiolus filled with tumor. The scala tympani of the lower coil was also filled with tumor; above this there was exudate. The nerve canals and vestibule were filled with tumor.

The microscopic examination of the labyrinthine nerve endings in cases of tumor of the nervus octavus is of interest in connection with the question of secondary degeneration of sensory nerve endings after they have been cut off from their ganglia. Wittmaack has cut the seventh and eighth nerves in animals before entrance into the internal meatus and has produced degenerative changes in the cochlear nerve ending in the labyrinth. These results are in conflict with the hitherto accepted idea regarding secondary degenerations. Waller's law states that after supraganglionary division of a sensory nerve there is no degeneration of the peripheral neurone. In cases of compression of the eighth nerve by neurofibroma Panse, and also Alexander, found atrophy of the spiral ganglion and loss of the sensory cells of Corti's organ. These changes may, however, have been due to compression of the internal auditory artery.

Case 1.—M. McQ., female, aged 75, was admitted to a poorhouse hosiptal on April 30, 1909, complaining of dizziness, deafness and noises in the left ear, of five years' duration. She could not walk without assistance. Rombergism marked. No note as to nystagmus. Patient has well marked arteriosclerosis.

Functional examination: C_{32} and C_{64} not heard by either ear. C_{128}, C_{256}, C_{512} and C_{1024} are all heard faintly by the right ear (side of tumor) but not by the left. C_{2048} not heard by either ear. Schwabach shortened. Galton's whistle not heard at all. Vestibular apparatus not tested. December 15, 1909: Patient can walk without help; deafness as before. Vision very bad. October, 1910: Dizziness worse; patient fell. January, 1911: Patient better. June, 1911: Indefinite headache. January, 1912: Patient completely deaf and bedridden. October, 1912: Sudden paralysis of right arm, leg and face. Death. Postmortem showed a tumor the size of a cherry involving the

eighth nerve on the right side. Gelatinous fluid present in air cells of mastoid on both sides.

Examination of right ear (side of acousticus tumor) : Mucosa of tympanic cavity and antrum slightly thickened. Delicate connective (?) tissue, very like tumor tissue, is seen in the scala tympani of the cochlea (Figs. 86, 87 and 88). There are large vessels in this tissue. The modiolus and spiral canal are infiltrated with the new connective or neuroglia (?) tissue (Figs. 86 and 87). In the lower part of the basal coil Corti's organ has quite disappeared (Fig. 87). In the upper part, Corti's organ is recognizable, and there are some ganglion cells left (Fig. 86). There is new bone formation in the scala tympani of the middle coil and in the basal coil (Figs. 86 and 87). The neuroepithelium of the utricle and saccule is well formed and the otolith membrane is present. The crista of the superior canal is atrophied when compared with the well formed cristæ of the lateral and posterior canals. New connective tissue formation is seen in the perilymph space of all the canals toward the convexity. The internal meatus is dilated and its posterior margin eroded by tumor tissue (Fig. 88). The tumor did not reach the fundus. The tumor, which shows large blood spaces, is composed of gliomatous or myxomatous tissue.

Examination of the left ear: The basal coil of the cochlea contains a considerable quantity of new bone and connective tissue (?) formation (Fig. 89). The scala vestibuli also shows new connective tissue (?) and bone formation. The scala media is greatly dilated. Corti's organ is present in all coils, and there is only slight atrophy of the spiral ganglion cells. Canals as on right side. The cochlear nerve, as is usual in old people, shows well marked corpora amylacia but is otherwise normal. The vestibular nerve is very atrophic.

The interesting thing in this case is the presence on both sides of changes which are usually associated with the presence of an old labyrinthitis. There is, however, very little evidence of middle ear suppuration. It is possible that the labyrinth has been infected from the meninges, as has been suggested by Siebenmann and Nager. Lastly, the presence of a tumor of the eighth nerve on the right side suggests that the condition of the inner ears may be due to an end stage of "choked

labyrinth." When the writer saw the patient in 1909 (three years before death) she could hear a little with the right ear (side of the tumor) but not at all with the left ear. This fact does not support the view that the changes in the two labyrinths were the result of the tumor in the right eighth nerve, although, on other grounds, this appears to be the most probable explanation.

Case 2.—A. T., female, aged 25, was an inmate of a hospital for incurables. Clinical details of the case are almost entirely wanting. The patient's face was very expressionless, and she was mentally dull and bedridden. She was blind and had double optic neuritis.

Postmortem examination: Outer (cranial) surface of dura, over almost its entire extent, was covered with small pedunculated tumors which varied in size from a hayseed to a split pea. On removal of the dura the inner surface of the cranial bones appeared as if deeply pitted by smallpox. In the region of the right internal auditory meatus and angle between the pons and cerebellum there was a tumor mass about the size of a walnut.

Examination of right ear: The tumor is eroding the posterior surface of the petrous bone (Figs. 90 and 91). The spiral ganglion in the lower part of the basal coil is infiltrated with tumor tissue and the ganglion cells have disappeared. The scala tympani is occupied by tissue resembling that of the tumor (Figs. 91 and 92). Internal to the round window membrane there is some new bone formation (Fig. 92). The perilymphatic aqueduct is filled with tumor. Corti's organ is unrecognizable in the lower part of the basal coil, although the patient was only 25 years old. In the middle and apical coils Corti's organ is fairly well formed. The neuroepithelium of the saccule and utricle is normal. The cristæ of the canals are practically normal. The internal meatus is greatly dilated by the tumor, especially at its inner end (Fig. 91). No giant cells are present, although the bone has been eroded. There is no appearance in this case of any otitis media. The condition of the dura mater and of the cerebral surface of the cranial bones was very interesting. The outer surface of the dura showed numerous small and rather pedunculated swellings which had eroded the inner or cranial surface of the

bones which form the "brain case" (Fig. 93). Cushing holds
that these small swellings on the dura are hypertrophied arach-
noid villi or Pacchionian bodies. If Cushing is correct in this
view, one would expect the swellings to be present in other
cases with increased intracranial pressure—e. g., in other va-
rieties of intracranial tumor. Professor Shennan, however, has
informed the writer that such is not the case. Professors Lor-
rain Smith and Theodore Shennan, Dr. J. W. Dawson and
Mr. Richard Muir have kindly examined the sections of these
projections from the dura and have given their opinions that
the tumors are composed of nerve tissue similar to that seen
in gliomata. Key holds that the protrusions are small glio-
matous "metastases" in the arachnoid. Without adopting the
idea of metastasis or secondary deposits of glioma due to blood
infection, it may well be that the small swellings in the brain
membranes are due to overgrowth of glia tissue—in other
words, that they are caused by a diffuse gliomatosis.

Case 3.—W. H., male, aged 43, had been deaf in the right
ear for six years but was otherwise quite well till two years
ago, when he was operated on for appendicitis. Soon after
he began to complain of dizziness and unsteady gait and of
occasional vomiting. Patient stated that he tended to stagger
to the right side. He also noticed weakness of the right side
when fatigued and right sided headache. A little later diplo-
pia was noted. Examination by Professor Bramwell showed
nystagmus on looking to the right and left, marked deafness
in the right ear. The grasp of the right hand was 85, while
that of the left was 120. (The patient was a right handed
man.) The finger-nose test was badly performed on the right
side. Ophthalmoscopic examination showed distinct optic
neuritis. The right knee jerk was more active than the left.
Professor Bramwell was of opinion that the hemiplegia did
not present any pyramidal characteristics and was probably
due to disturbance of the cerebellum on the right side. He
thought that the patient's condition was probably caused by an
extracerebellar growth originating in the eighth nerve. Later,
a change in the position of the head was noted. It was held
slightly depressed to the left, with the chin pointing to the
right—the position assumed by the head of a monkey after one
lateral lobe of the cerebellum has been removed. This posi-

tion could not be regarded as of localizing value, since the head is sometimes depressed towards the side of the tumor and sometimes toward the opposite side.

Otologic Examination: Cochlear apparatus: The loud voice was not heard by the right ear when the noise box was placed in the left ear. Weber lateralized to the left (good) ear. Rinne "infinite negative" on right side. Vestibular apparatus: Marked nystagmus to the right, less marked to the left; there was also distinct nystagmus to the left on looking up. Pointing test normal. No Rombergism. On cold syringing of the right ear there was no increase in the nystagmus to the left. Galvanic reaction: With the positive pole applied to the right ear it required 14 ma. to make the patient fall to the right. The same, however, prevailed on the left side. There was no facial paralysis and the patient could appreciate salt on the anterior part of the tongue on both sides.

X-ray examination: This appeared to show that the internal auditory meatus was larger on the affected than on the normal side.

Operation by a general surgeon. The tumor, however, was not reached, even with the finger.

Postmortem: Tumor the size of a large walnut in the cerebellopontine angle on the right side.

Examination of right ear: The growth has eroded the bone above and in front of the cochlea (Figs. 94 and 97). The lamellar bone has yielded much more easily to erosion than the cartilage bone. Tumor tissue intervenes between the internal carotid artery and the cochlear capsule (Fig. 97). The new growth indeed comes right up to the carotid canal. The tumor appears rather to have compressed the cochlear nerve than to have infiltrated it (Fig. 95). The basal coil of the cochlea shows both scala tympani and scala vestibuli full of fibrinous exudate (Figs. 95 and 96). Reissner's membrane is bulged upwards. Corti's organ is not well formed in the lower part of the basal coil. The cochlear ganglion cells appear to be shrunken. The utricle and saccule are dilated (Fig. 94) but the neuroepithelium is normal. In the perilymph space of the vestibule, between the saccule and the oval window, granular fibrinous material is present (Fig. 94). The canals show exudate in both endo- and peri-lymph spaces, but

the cristæ and cupulæ are normal. The internal meatus is
enormously enlarged by the tumor (Figs. 94 and 97). The
whole anterior wall of the internal meatus has been eroded and
the dural lining is present only in the fundus. The tumor
tissue invades the saccus endolymphaticus. This may account
for the dilated condition of the utricle and saccule.

Remarks.—In this case the condition of the labyrinth dif-
fered from that seen in Cases 1 and 2. There was no new
connective tissue or bone formation. Although deafness in
the affected ear had been present for six years, it is possible
that Case 3 was of comparatively short duration when con-
trasted with Cases 1 and 2. If this is so, we may look on the
fibrinous exudate as the early stage of "choked labyrinth,"
while the formation of new connective tissue and bone repre-
sents the late stage. In all three cases there was definite evi-
dence of degeneration of the nervous structures of the cochlea,
whereas the vestibular end organs were comparatively well
formed.

Fig. 1.—Coronal section No. 21. 1, Tensor tympani; 2, eustachian
tube; 3, floor of middle cranial fossa; 4, eighth nerve in the internal
meatus; 5, posterior surface of petrous bone; 6, glossopharyngeal
nerve and opening of aqueduct of cochlea; 7, marrow spaces.

Fig. 2.—Coronal section No. 97. 1, Tensor tympani; 2, apical coil
of cochlea; 3, middle coil; 4, cochlear nerve in internal meatus; 5,
cranial end of aqueduct of cochlea (perilymph); 6, basal coil of
cochlea; 7, carotid canal; 8, tubal portion of tympanic cavity.

Fig. 3.—Coronal section No. 127. 1, Tensor tympani; 2, helico-
trema; 3, canal for great superficial petrosal nerve; 4, modiolus; 5,
facial nerve; 6, cochlear aqueduct; 7, edge of cartilage bone capsule
of cochlea; 8, carotid canal; 9, tubal cells; 10, tubal part of tympanic
cavity.

Fig. 4.—Coronal section No. 175. 1, Tensor tympani; 2, facial
nerve passing above cochlea; 3, vestibular ganglion; 4, cochlear
aqueduct and vein accompanying it; 5, jugular bulb; 6, air cells in
floor of tympanic cavity; 7, tympanic cavity.
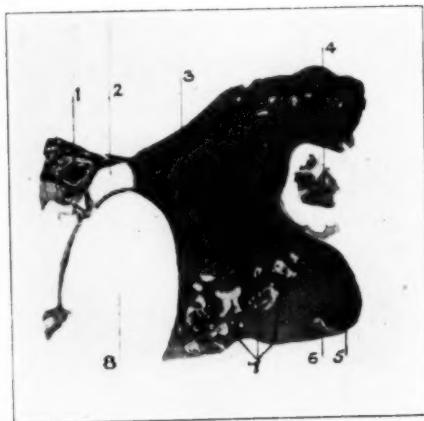
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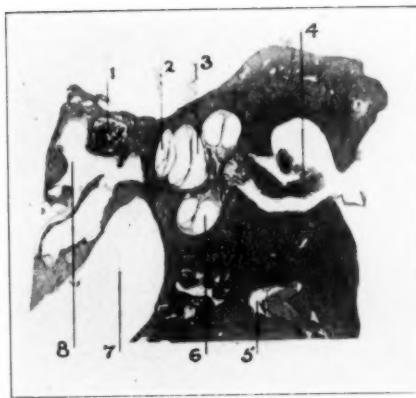
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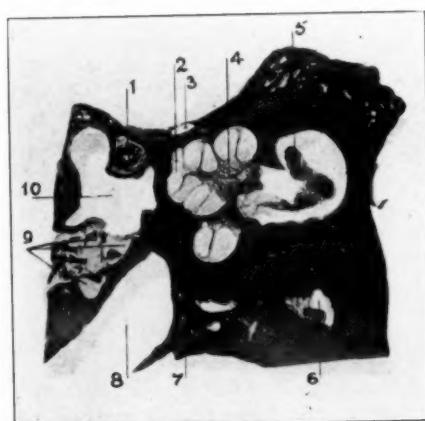
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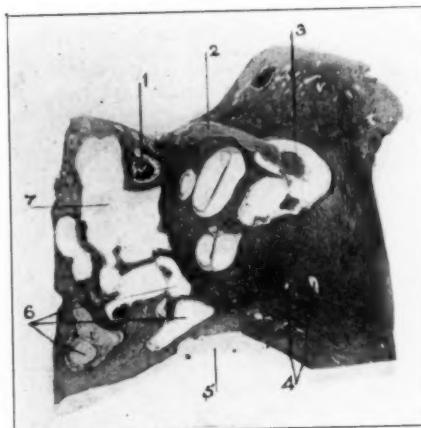
Fig. 4.

Fig. 5.—Coronal section No. 244. 1, Anterior part of epitympanic cavity; 2, facial nerve; 3, anterior part of utricle; 4, cochlear opening of perilymphatic aqueduct; 5, jugular bulb; 6, tympanic air cells; 7, tympanic membrane; 8, external meatus; 9, tympanic cavity; 10, sacculus; 11, tensor tympani.

Fig. 6.—Coronal section No. 295. 1, Tensor tympani; 2, facial nerve; 3, utricle; 4, ampulla of superior canal with crista; 5, opening of crus commune; 6, superior canal; 7, sinus of posterior canal with crista quarta; 8, niche of round window; 9, jugular bulb; 10, promontory; 11, external meatus; 12, handle of malleus attached to drumhead; 13, head, and 14, footplate of stapes.

Fig. 7.—Coronal section No. 320. 1, External attic; 2, head of malleus; 3, facial nerve; 4, two ends of lateral canal; 5, superior canal; 6, fossa subarcuata; 7, aqueduct of vestibule; 8, two ends of posterior vertical canal; 9, jugular bulb; 10, tympanic cavity; 11, external meatus; 12, tympanic membrane; 13, stapes; 14, Prussac's space.

Fig 8.—Coronal section No. 385. 1, Head of malleus; 2, body of incus; 3, external canal; 4, air cells; 5, endolymphatic sac; 6, two ends of posterior canal; 7, jugular bulb; 8, drumhead; 9, sinus tympani.
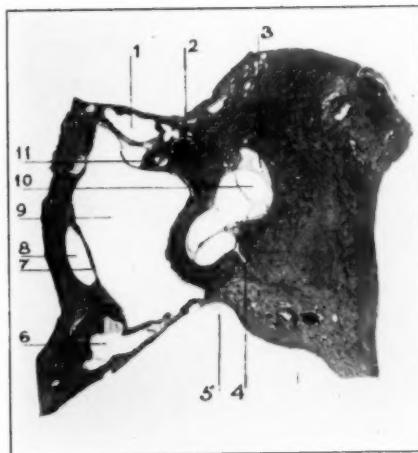
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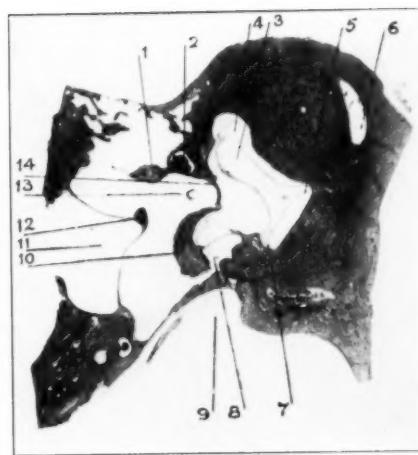
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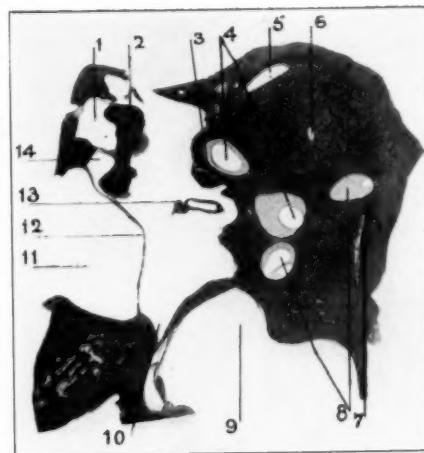
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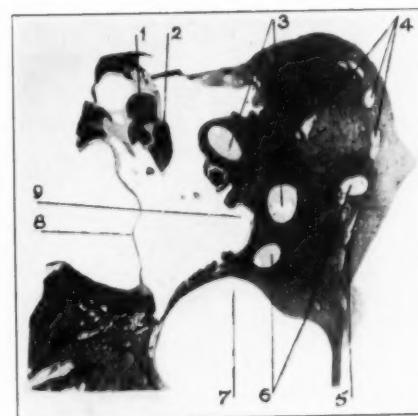
Fig. 8.

Fig. 9.—Coronal section No. 450. 1, Posterior part of drumhead; 2, external acoustic meatus; 3, outer wall of aditus; 4, short process of incus; 5, roof of tympanic antrum; 6, prominence of lateral canal on inner wall of aditus; 7, perilymph space of lateral canal; 8, air cells on inner wall of tympanic antrum; 9, two ends of posterior vertical canal; 10, saccus endolymphaticus; 11, air cells below labyrinth; 12, sinus tympani; 13, stapedius muscle; 14, facial nerve.

Fig. 10.—Coronal section No. 523. 1, Roof of mastoid antrum; 2, cavity of antrum; 3, posterior canal; 4, air cells; 5, saccus endolymphaticus; 6, air cells; 7, sinus tympani; 8, facial nerve; 9, chorda tympani; 10, external meatus; 11, border air cells overhanging external meatus.

Fig. 11.—Horizontal section No. 140. 1, Outer wall of attic; 2, body of incus; 3, head of malleus; 4, anterior ligament (remains of Meckel's cartilage); 5, inner attic; 6, geniculate ganglion; 7, basal coil of cochlea; 8, ampullary and smooth ends of superior vertical canal; 9, fossa subarcuata; 11, prominence of lateral canal.

Fig. 12.—Horizontal section No. 155. 1, Outer wall of attic; 2, malleus; 3, tendon of tensor tympani; 4, facial nerve; 5, middle coil of cochlea; 6, basal coil; 7, internal meatus with facial nerve to right and vestibular nerve to left; 8, ampulla of lateral canal, with crista; the short line points to the superior vertical canal; 9, fossa subarcuata; 10, tympanic antrum.
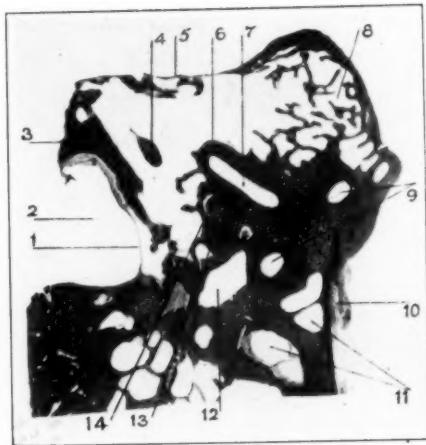
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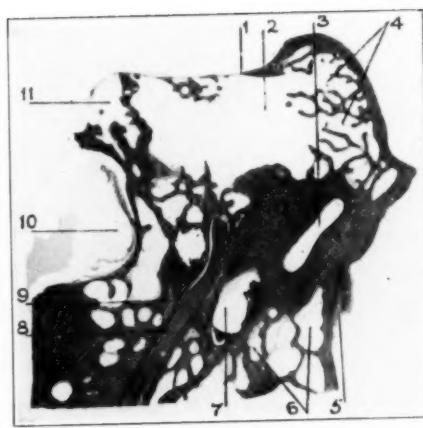
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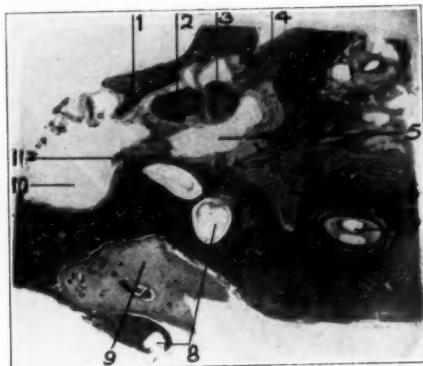
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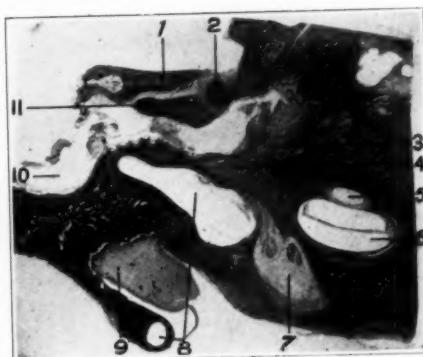
Fig. 12.

Fig. 13.—Horizontal section No. 205. 1, Footplate of stapes; 2, posterior part of annulus tympanicus; 3, handle of malleus; 4, tympanic cavity containing exudate usually found in the newborn child; 5, helicotrema of cochlea; 6, anterior wall of internal auditory meatus; 7, internal meatus with nerves; 8, saccule; 9, utricle; 10, crus commune; 11, lateral canal.

Fig. 14.—Horizontal section No. 255. 1, Facial nerve; 2, long process of incus; 3, head of stapes; 4, handle of malleus; 5, eustachian tube; 6, basal coil of cochlea; 7, internal meatus; 8, saccule; 9, footplate of stapes; 10, junction of crus commune with utricle; 11, smooth end of lateral canal opening into vestibule; 12, posterior canal.

Fig. 15.—Horizontal section No. 320. 1, Facial nerve; 2, handle of malleus with drumhead; 3, anterior part of annulus tympanicus; 4, eustachian tube; 5, carotid artery with accompanying veins and nerves; 6, basal coil of cochlea; 7, internal meatus; 8, junction between perilymph space of vestibule and scala vestibuli of cochlea; 9, vestibule; 10, lower part of utricle; 11, groove for saccus endolymphaticus; 12, posterior vertical canal; 13, sinus tympani; 14, stapedius muscle.

Fig. 16.—Horizontal section No. 350. 1, Facial nerve; 2, stapedius; 3, niche of round window; 4, drumhead below level of malleus; 5, scala vestibuli of basal coil; 6, tympanic opening of eustachian tube; 7, carotid canal; 8, scala tympani; 9, membrane of round window; 10 and 11, two ends of posterior vertical canal.
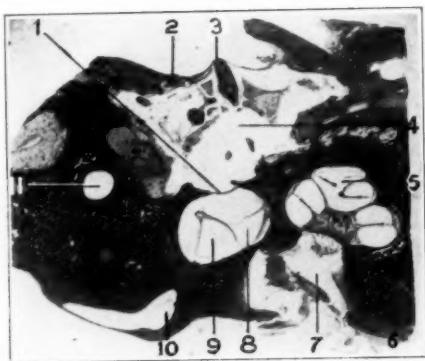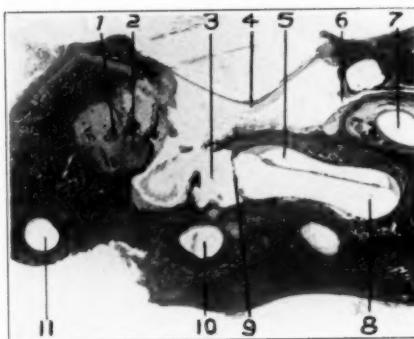
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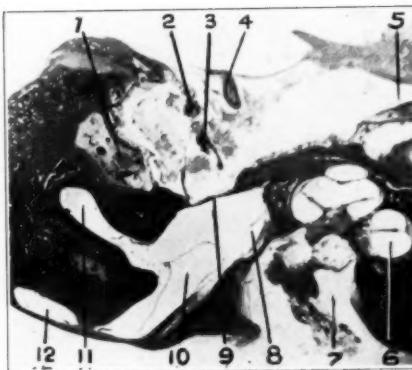
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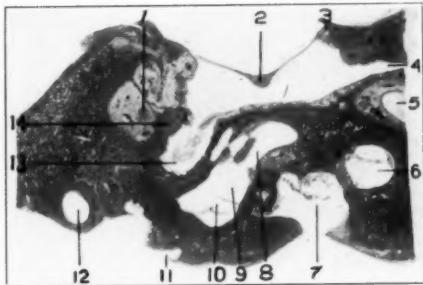
Fig. 15.

Fig. 16.

Fig. 17.—Case 1. Fracture of cranial base without fracture of labyrinth capsule. J. P., male, aged 54. Horizontal section through right ear, No. 122. 1, Footplate of stapes; 2, malleus; 3, hemorrhage; 4, incus; 5, vestibule; 6, posterior canal; 7, region of saccus endolymphaticus; 8, internal meatus; 9, basal coil of cochlea.

Fig. 18.—Case 1. Section through tympanic cavity. 1, Hemorrhage posterior to malleus and incus; 2 and 3, malleus; 4, incus; 5, stapes; 6, hemorrhage around facial canal; 7, facial nerve.

Fig. 19.—Case 1. Horizontal section through facial nerve and upper part of internal meatus. 1, Facial nerve; 2, geniculate ganglion; 3, basal coil of cochlea; 4, facial nerve showing ganglion cells along its course; 5, vestibular nerve with ganglion cells; 6, upper part of vestibule; 7, hemorrhage along branch of vestibular nerve to crista of lateral canal.

Fig. 20.—Case 1. Horizontal section No. 170. 1, Hemorrhage in modiolus; 2, blood in perilymphatic aqueduct; 3, cochlear opening of the aqueduct.

Fig. 17.

Fig. 18.
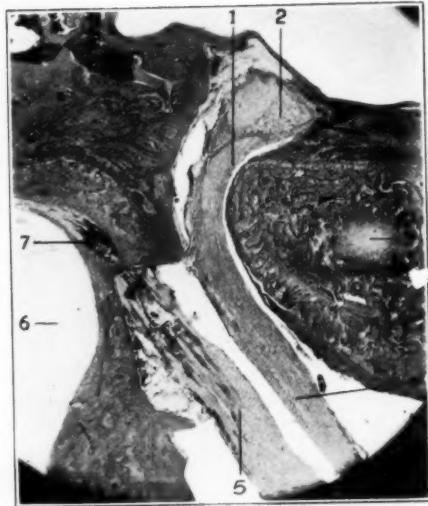
Fig. 19.

Fig. 20.

Fig. 21.—Case 2. Horizontal section through right ear. 1, Line of fracture on inner wall of attic—below this in the photograph there is blood in both peri- and endolymphatic spaces of the superior and lateral canals; 2, hemorrhage in endolymph space of lateral canal; 3, blood in air cell on inner wall of mastoid antrum; 4, line of fracture reaches a very large air cell, internal to the labyrinth; the line from 4 crosses the fossa subarcuata; 5, hemorrhages in the upper part of the basal coil of the cochlea.

Fig. 22.—Case 2. Horizontal section through right ear in the region of the oval window. 1, Fracture passes through footplate of the stapes; 2, lateral canal with blood in endo- and perilymph spaces; 3, hemorrhage in air cell internal to the labyrinth; 4, smooth end of superior canal free from blood; 5, fracture through vestibule from 1 and reaches 5; 6, hemorrhage in the vestibular nerve; 7, cochlear nerve with slight bleeding at the base of the modiolus; 8, basal coil of cochlea with blood in scala tympani and vestibule.

Fig. 23.—Case 2. Horizontal section through right ear in region of round window. 1, Line of fracture; 2, blood in convexity of posterior canal; 3, endolymphatic aqueduct free from hemorrhage; 4, ampulla of posterior canal with blood in it; 5, inner end of fracture reaches internal auditory meatus; 6, scala tympani of basal coil with hemorrhage; 7, cochlear canal contains blood.

Fig. 24.—Case 3. Vertical section. 1, Tensor tympani; 2, scala tympani of upper part of basal coil, filled with hemorrhagic exudate; 3, upper end of fracture; 4, the cochlear nerve surrounded by meningitis; 5, aqueduct of cochlea; 6 lower end of fracture; 7, basal coil filled with exudate; 8, exudate in tubal portion of tympanic cavity.
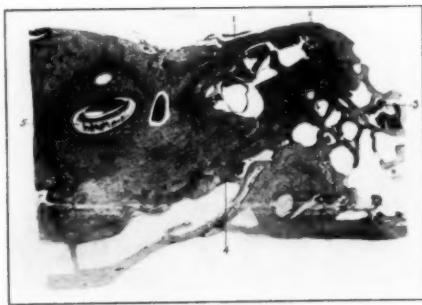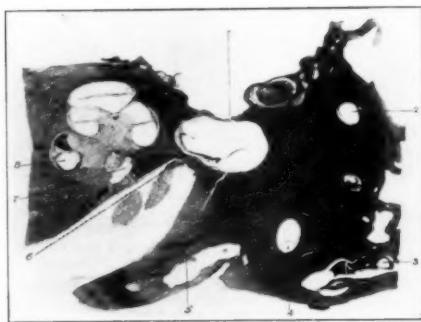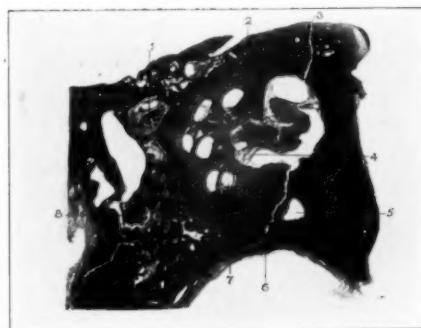
Fig. 21.

Fig. 22.

Fig. 23.

Fig. 24.

Fig. 25.—Case 3. Vertical section. 1, Facial nerve; 2, upper end of fracture, which passes into vestibule; 3, hemorrhagic purulent exudate in vestibule; 4, fracture through bony spiral lamina; 5, exudate in scala tympani; 6, lower end of fracture; 7, hemorrhagic exudate in tympanic cavity; 8, perforation of tympanic membrane.

Fig. 26.—Case 3. Vertical section. 1, Head of malleus; 2, fracture of tympanic roof; 3, superior vertical canal; 4, two ends of lateral canal; 5, ductus endolymphaticus; 6, two ends of posterior vertical canal; 7, exudate in tympanic cavity; 8 and 10, fracture of external meatus; 9, tympanic membrane below perforation.

Fig. 27.—Case 3. Vertical section. 1, Head of malleus; 2, exudate in attic; 3, two ends of lateral canal; 4, superior canal; 5, exudate in air cell internal to labyrinth; 6, two ends of posterior canal; 7, exudate in sinus tympani; 8, sinus tympani; 9, fracture of anterior wall of external meatus; 10, external meatus; 11, perforation of drumhead; 12, long process of incus.

Fig. 28.—Case 4. Vertical section. 1, Superior canal; 2, position which should be occupied by incus; 3 head of malleus; 4, exudate in tympanum; 5, stapedius muscle; 6, two ends of posterior canal; 7, two ends of lateral canal.
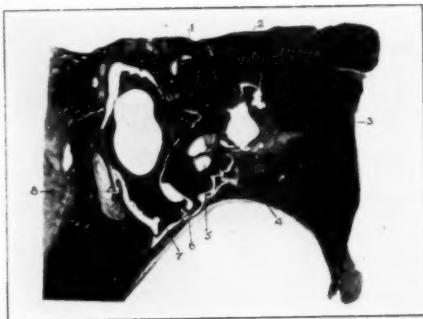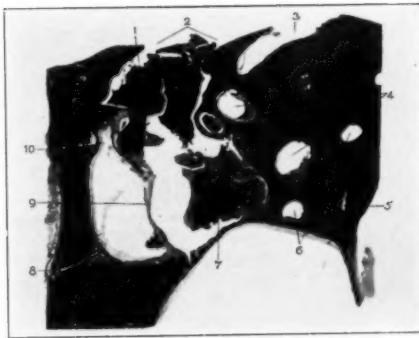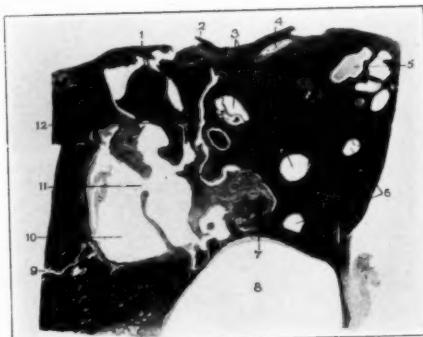
Fig. 25.

Fig. 26.

Fig. 27.

Fig. 28.

Fig. 29.—Case 4. Vertical section. 1, Connective tissue in gap left by old fracture; 2, long process of incus; 3, articular surface of incus; 4, tip of short process of incus which has remained attached to floor of aditus while the rest of the bone became dislocated upwards and backwards into the antrum; 5, epidermic lining of external meatus peeling off; 6, exudate in air cells behind labyrinth; 7, air cells with inflamed lining membrane on inner well of antrum.

Fig. 30.—War Injury. Case 1. Direct injury to the ear by shrapnel, followed by suppurative otitis media, cerebellar abscess, etc. Horizontal section through attic of left ear. 1, Malleus dislocated off incus; 2, facial nerve; 3, ampulla of superior canal; 4, perilymph space of lateral canal; 5, body of incus—note the thickened, engorged and infiltrated mucosa of the attic surrounding the ossicles.

Fig. 31.—Case 1. Section through tympanic cavity, showing: 1, perforation of drumhead; 2, greatly thickened tympanic membrane; 3, handle of malleus separated from the drumhead; 4, long process of incus, which is eroded; 5, fracture of stapes; 6, facial canal—the nerve is absent (artefact); 7, corda tympani nerve; 8, posterior part of drumhead.

Fig. 32.—Case 1. Horizontal section through region of oval window, showing 1, thickened and infiltrated drumhead; 2 handle of malleus separated from drumhead; 3, part of tympanic membrane carried inward with drumhead; 4, basal coil of cochlea with hemorrhage; 5, posterior part of annular ligament, infiltrated; 6, joint between incus and stapes—note the pus in the tympanic cavity.
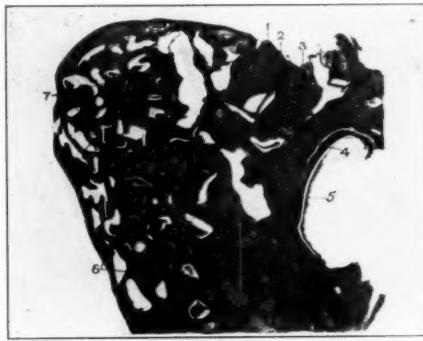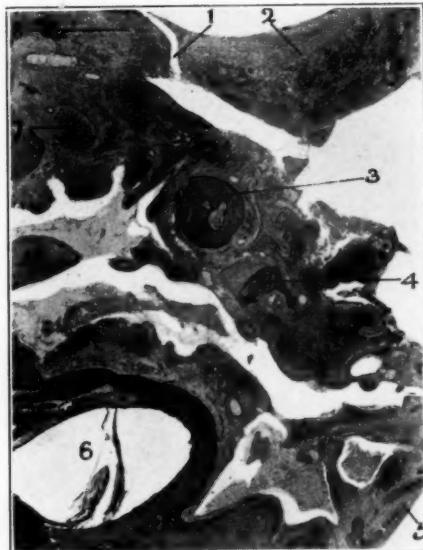
Fig. 29.
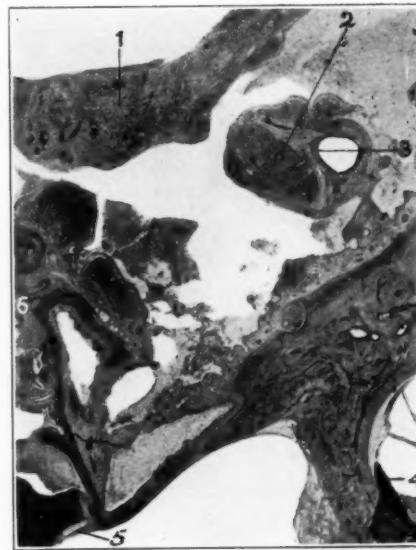

Fig. 30.

Fig. 31.

Fig. 32.

Fig. 33.—Case 1. Horizontal section through region of round window. 1, Engorged and infiltrated mucosa of promontory; 2, cochlear nerve in internal meatus, with meningitis; 3, hemorrhage in scala tympani; 4, hemorrhage in perilymph space of ampulla of posterior canal; 5, fracture between sinus tympani and crista of posterior canal; 6, exudate in niche of round window.

Fig. 34.—Case 2. Bullet wound of frontoparietal region, followed by intracranial hemorrhage. No direct injury to ear. Vertical section through cochlea, showing 1, hemorrhage in fundus of internal meatus around branches of cochlear nerve to the basal coil.

Fig. 35.—Case 2. Vertical section through vestibule and intravestibular part of cochlea. 1, Hemorrhage around nerve above vestibule; 2, facial nerve; 3, hemorrhage on inner wall of tympanum; 4, niche of oval window; 5, cochlear canal; 6, exudate in cochlear opening of perilymphatic aqueduct; 7, hemorrhage around nerve to ampulla of posterior canal.

Fig. 36.—Case 3. High explosive shell injury of ear. Horizontal section showing 1 and 2, everted edges of rupture in drumhead; 3, handle of malleus; 4, facial nerve with stapedius to left—the sinus aympani lies below; 5, posterior canal; 6, utricle; 7, internal meatus; 8, carotid canal; 9, opening of eustachian tube.
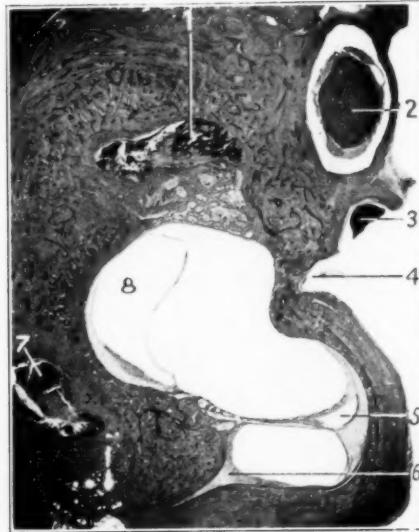
Fig. 33.
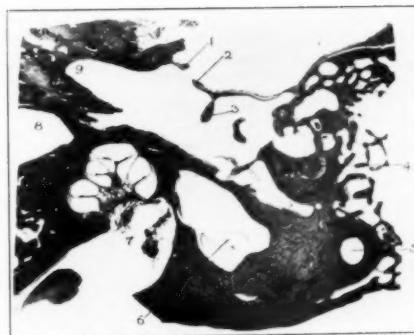

Fig. 34.

Fig. 35.

Fig. 36.

Fig. 37.—Case 4. Injury of ear due to explosion of rifle grenade. Horizontal section No. 125. 1, Facial nerve; 2, utricle; 3, detached otolith membrane of utricle; 4, ampullary end of lateral canal.

Fig. 38.—Case 4. Horizontal section through oval window, showing, 1, footplate of stapes; 2, neuroepithelium of saccule; 3, detached otolith membrane of saccule.

Fig. 39.—Case 4. Horizontal section through region of round window. 1, Rupture of drumhead; 2, scala tympani of basal coil of cochlea; 3, internal meatus with hemorrhage.

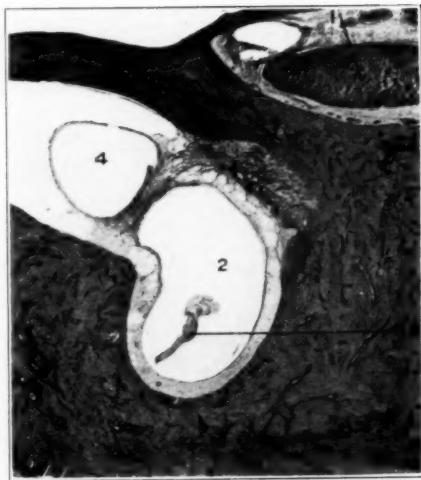Fig. 40.—Case 4. Shows displacement of crista of lateral canal
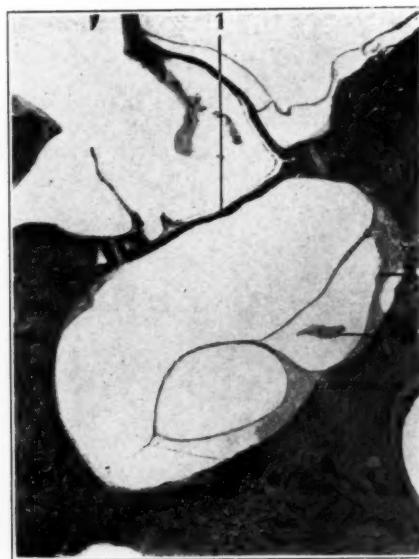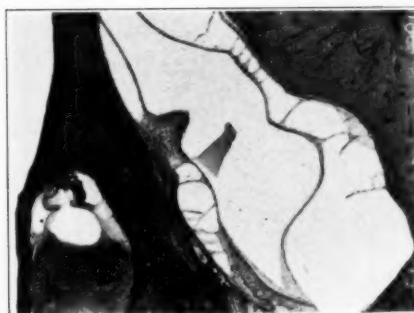
Fig. 37.

Fig. 38.

Fig. 39.

Fig. 40.

Fig. 41.—Case 6. High explosive shell injury of ear. Horizontal section through region of oval window. 1. Posterior part of rupture of drumhead; 2, handle of malleus with remains of drumhead attached; to the right the anterior part of the rupture is seen; 3, long process of incus; 4, hemorrhage in hollow of stapes; 5, facial canal; 6, chorda tympani nerve.

Fig. 42.—George Dick, aged 4. Chronic middle ear suppuration; erosion of lateral canal; cerebellar abscess. 1, Cholesteatoma covering prominence of lateral canal; 2, erosion of bone; 3, endolymph space; 4, perilymph space.

Fig. 43.—Donald Henry, aged 23. Chronic middle ear suppuration, right ear; temporosphenoidal abscess; meningitis. 1, Fistula in lateral canal; 2, perilymph space of lateral canal; 3, facial nerve; 4, erosion of bone.

Fig. 44.—Annie Bruce, aged 15. Chronic middle ear suppuration, right ear; cholesteatoma; latent labyrinthitis; perisinus abscess; sinus thrombosis. 1, Endosteum lining lateral canal; 2, endolymph space containing exudate; 3, granulation tissue in perilymph space; 4, facial nerve.
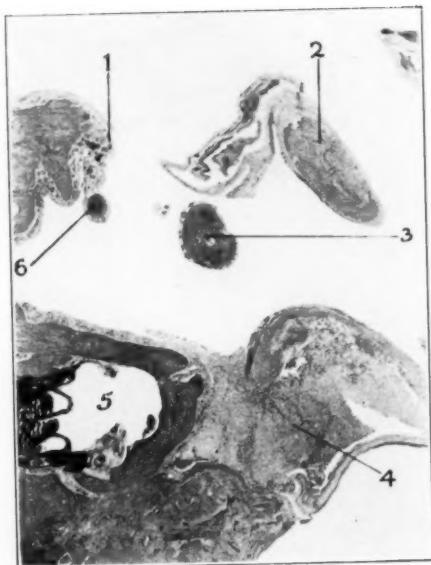
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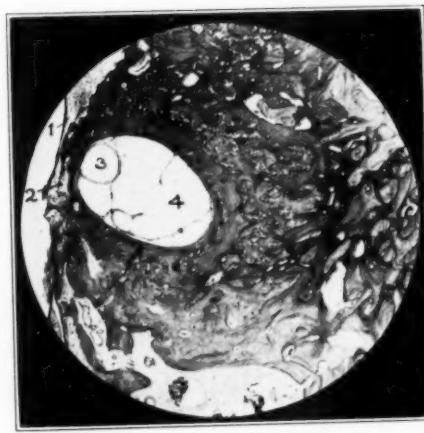
Fig. 42.

Fig. 43.
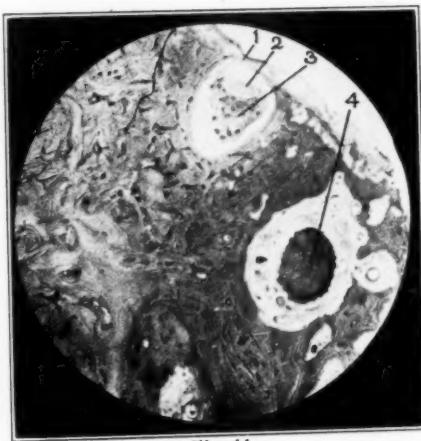
Fig. 44.

Fig. 45.—John Fisher, aged 11. Chronic middle ear suppuration, right ear; cholesteatoma; meningitis. 1, Prominence of lateral semicircular canal; 2 coagulated lymph in perilymph space of lateral canal (serous meningitis?); 3, smooth end of lateral canal; 4, facial nerve.

Fig. 46.—Same specimen as Fig. 45 under higher power. 1, Endolymph space, normal; 2, perilymph space containing coagulated fibrin.

Fig. 47.—John Johnson, aged 12. Acute middle ear suppuration (bilateral); vomiting; restlessness, with meningitic cry followed by coma. Right ear, showing hemorrhages into labyrinth but no labyrinthitis. 1, Tensor tympani; 2, purulent exudate in tympanum; 3, malleus; 4, incus; 5, exudate; 6, facial nerve—below the facial nerve there is hemorrhage into the perilymph space of the vestibule; 7, superior canal; 8, internal meatus with meningitis; 9, basal coil of cochlea with hemorrhage in scala vestibuli; 10, carotid canal.

Fig. 48.—John Johnson right ear. Basal coil of cochlea. 1, Hemorrhage in cochlear canal; 2, scala vestibuli; 3, spiral ganglion; 4, hemorrhage along nerve in bony spiral lamina; 5, scala tympani.

Fig. 45.

Fig. 46.
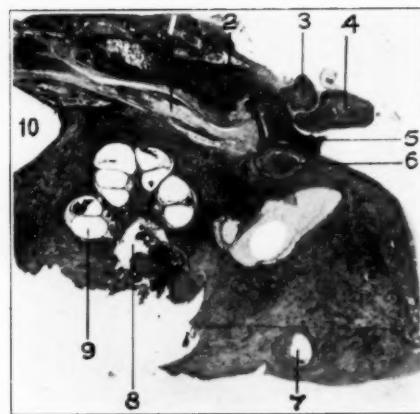
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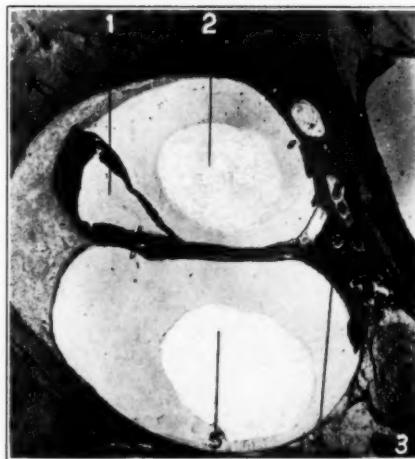
Fig. 48.

Fig. 49.—John Johnson, right ear. Horizontal section through oval window. 1, Hemorrhage external to sacculus; 2, pus in hollow of stapes; 3, footplate of stapes; 4 and 6, hemorrhage in vestibule; 5, utricle; 7, ductus endolymphaticus; 8, saccule.

Fig. 50.—John Johnson, right ear. Horizontal section through lower part of tympanum and labyrinth. 1, Pus in tympanic cavity; 2, external meatus; 3, joint between incus and stapes; 4, facial nerve; 5, posterior canal; 6, aqueduct of vestibule; 7, junction of perilymph space of vestibule with scala vestibuli; 8, hemorrhage in basal coil of cochlea.

Fig. 51.—John Johnson, right ear. 1, Scala tympani; 2, hemorrhage internal to round window membrane; 3, niche of round window lined by swollen mucosa; 4, membrane of round window; 5, pus in cochlear opening of perilymphatic aqueduct; 6, vein accompanying perilymphatic aqueduct.

Fig. 52.—John Johnson, left ear. On this side purulent labyrinthitis was present in addition to middle ear suppuration. Horizontal section through region of oval window. 1, External meatus; 2, incus; 3, malleus; 4, facial nerve; 5, swollen mucosa of tympanum; 6, basal coil of cochlea containing pus; 7, internal meatus; 8, pus in vestibule; 9, utricle; 10, opening of lateral canal into vestibule; 11, lateral canal; 12, posterior canal.

Fig. 49.
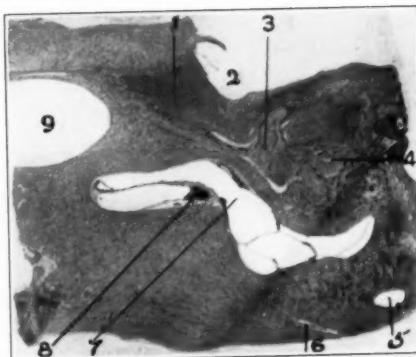
Fig. 50.

Fig. 51.
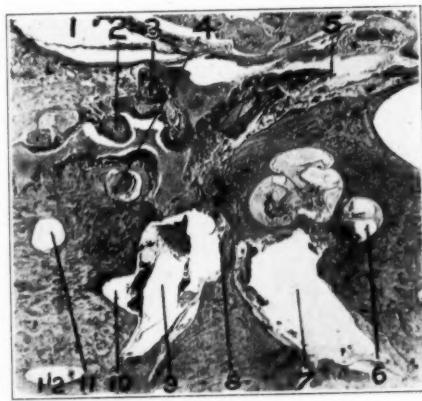
Fig. 52.

Fig. 53.—John Johnson, left ear. Horizontal section through oval window. 1, Swollen mucosa over facial canal; 2, pus in tympanic cavity; 3, footplate of stapes—notice the pus entering the vestibule at both edges of the footplate; 4, posterior crus of stapes; 5, internal meatus with meningitis; 6, ruptured saccule; 7 and 8, pus in vestibule; 9, utricle.

Fig. 54.—John Johnson, left ear. Section through drumhead, showing formation of perforation. 1, Chorda tympani; 2, external meatus; 3 pus in tympanum bursting through fibrous layer of drumhead; 4, desquamated epithelium of drumhead; 5, fibrous layer of drumhead, 6, promontory: 7, mucous membrane layer of drumhead; 8, pus in tympanum.

Fig. 55.—John Johnson, left ear. Horizontal section through region of round window. 1, Facial nerve; 2, stapedius; 3, external meatus; 4, swollen drumhead; 5, carotid canal; 6, basal coil of cochlea full of pus; 7, posterior part of membrane of round window; 8 and 9, two ends of posterior vertical canal.

Fig. 56.—John Johnson, left ear. Section through region of round window. 1, Pus in tympanum; 2, swollen mucosa of promontory; 3, pus in scala tympani; 4, vein accompanying aqueduct of cochlea; 5, cochlear opening of perilymphatic aqueduct; 6, pus in niche of round window—note that between 3 and 6 the membrane of the round window is disintegrated; 7, ampullary end of posterior canal.

Fig. 53.
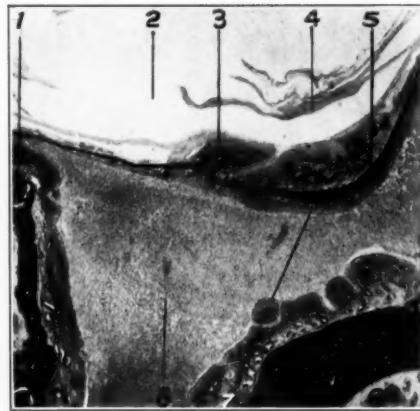
Fig. 54.
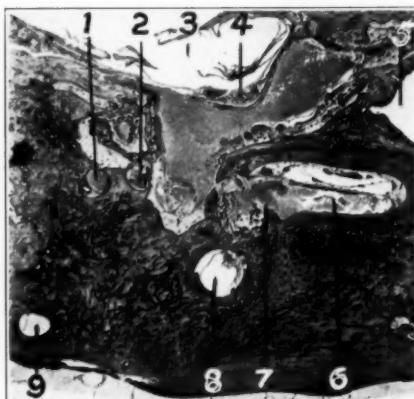
Fig. 55.

Fig. 56.

Fig. 57.—Mrs. Carson, aged 20. Chronic middle ear suppuration, right ear; vomiting, shivering and headache; giddiness; facial paralysis and third degree nystagmus to left. Vertical transverse section through oval window. 1, Facial canal; 2, thickened mucosa in niche of oval window; 3, pus in perilymph space of vestibule and scala vestibuli; 4, exudate in scala tympani; 5, footplate of stapes displaced into vestibule.

Fig. 58.—Mrs. Carson. Vertical transverse section through oval window. 1, Ampullary end of superior canal; 2, facial nerve; 3, oval window, empty; 4, thickened mucosa of round window; 5, ampullary end of posterior canal containing pus; 6, opening of crus commune.

Fig. 59.—Mrs. Carson. Section through basal coil of cochlea. 1, Fibrinous exudate in scala vestibuli; 2, dilated cochlear canal; 3, spiral ligament; 4, fibrinous exudate in scala tympani; 5, Corti's organ; 6, nerve in bony spiral lamina; 7, Reissner's membrane pulled inwards towards modiolus by contraction of exudate in scala vestibuli.

Fig. 60.—Thomas Gilmour, aged 17. Chronic middle ear suppuration, right; giddiness; death from cerebellar abscess. 1, Superior canal containing granulation tissue; 2, ampullary end of superior canal; 3, fistula in lateral canal; 4, facial nerve surrounded by granulation tissue; 5, polypus projecting into external meatus; 6, perforation of drumhead; 7, jugular bulb showing new connective tissue and bone formation; 8, ampullary end of posterior canal with pus; 9, lateral canal.
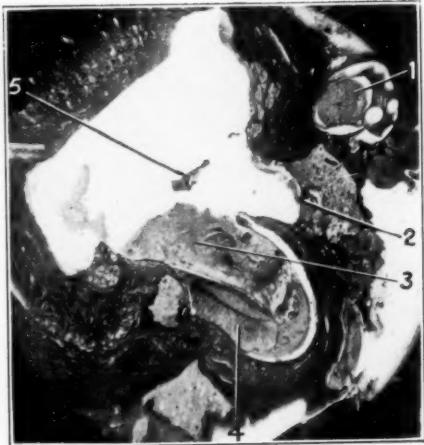
Fig. 57.

Fig. 58.
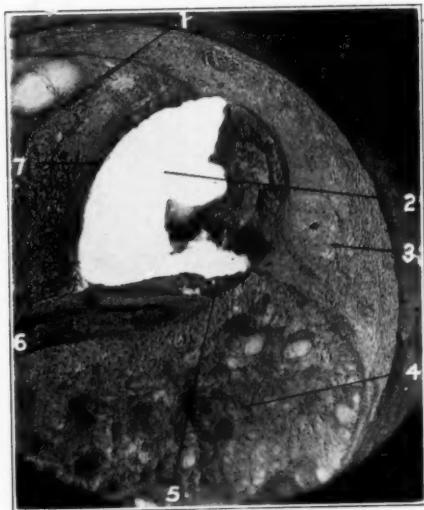
Fig. 59.

Fig. 60.

Fig. 61.—Thomas Gilmour. 1, Cholesteatoma in antrum; 2, polypus, which is originating in posterior cranial fossa; 3, stapedius; 4, lower part of posterior canal; 5, region of extradural abscess.

Fig. 62.—Christina Goodall, aged 6. Chronic middle ear suppuration, left ear; latent labyrinth suppuration; panlabyrinthitis; septicemia. Horizontal section through attic. 1, Outer attic; 2, polypus projecting from inner wall of attic; 3, cholesteatoma; 4, smooth end of superior canal; 5, ampullary end of superior canal, surrounded by area of granulation tissue; 6, basal coil of cochlea; 7, facial nerve.

Fig. 63.—Christina Goodall. Panlabyrinthitis. Horizontal section through upper part of vestibule and cochlea. 1, Osteomyelitis in promontory; 2, cholesteatoma in tympanum; 3, facial nerve; 4, lateral canal surrounded by granulation tissue; 5, cholesteatoma in mastoid; 6 and 7, engorged marrow spaces; 8, smooth end of superior canal containing pus; 9, pus in vestibule; 10, area of osteomyelitis; 11, vestibular nerve embedded in granulation tissue; 12, cochlear nerve; 13, basal coil of cochlea containing granulation tissue—note the dilatation of the cochlear canal.

Fig. 64.—Christina Goodall. Panlabyrinthitis. Section through lower part of vestibule and cochlea. 1, Cholesteatoma; 2, malleus; 3, incus; 4, lateral canal surrounded by granulation tissue (osteomyelitis); 5, crus commune in some condition; 6, footplate of stapes; 7, basal coil of cochlea filled by granulation tissue; 8, upper part of basal coil.
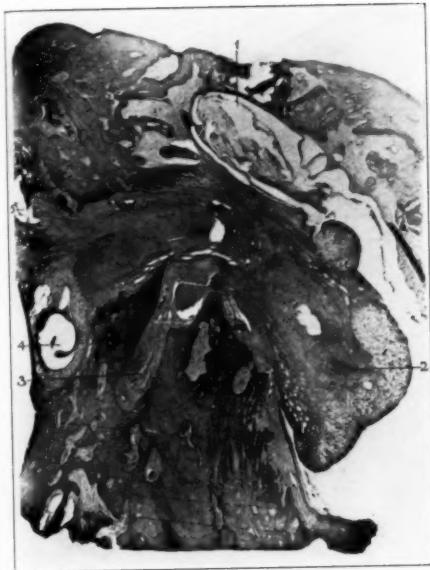
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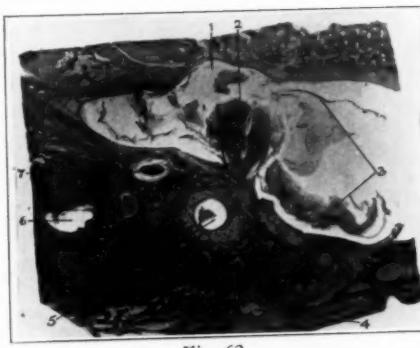
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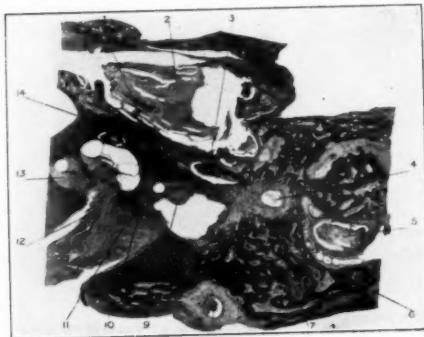
Fig. 63.

Fig. 64.

Fig. 65.—Christina Goodall. Panlabyrinthitis. Horizontal section through lower part of labyrinth. 1, New connective tissue in scala tympani internal to round window; 2, stapedius; 3 and 4, malleus and incus; 5, facial nerve; 6, opening of smooth end of lateral canal into vestibule—note the osteomyelitis around this and also the crus commune below it; 7, endolymphatic aqueduct; 8 and 9, areas of osteomyelitis; 10, new bone formation in scala tympani of basal coil; 11, carotid canal.

Fig. 66.—Walter Penman, aged 42. Chronic middle ear suppuration, left; giddiness; headache; insanity developed; death from endocarditis. Vertical transverse section through cochlea. 1, Internal meatus showing great thickening of dura and new fibrous tissue formation around nerves; 2, modiolus; 3, middle coil of cochlea with exudate; 4, tensor tympani; 5, tympanus; 6, carotid canal; 7, basal coil of cochlea filled up by new bone; 8, jugular bulb; 9, perilymphatic aqueduct.

Fig. 67.—Walter Penman. Vertical section through vestibule. 1, Extradural abscess between middle and posterior cranial fossae; 2, vestibule filled by connective tissue; 3, oval window; 4, facial nerve; 5, polypoid mucosa of tympanum; 6, malleus; 7, external meatus; 8, promontory; 9, niche of round window filled by connective tissue; 10, ampullary end of posterior canal; 11, jugular bulb.

Fig. 68.—William Newberry, aged 26. Chronic middle ear suppuration, left ear; cholesteatoma; sinus thrombosis; latent labyrinth suppuration; cerebellar abscess. Vertical transverse section through anterior part of cochlea. 1, Facial nerve; 2, internal meatus; 3, connective tissue in vestibule and basal part of cochlea; 4, region of round window; 5, promontory; 6, new bone formation.
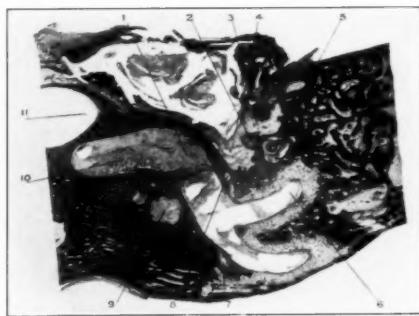
Fig. 65.
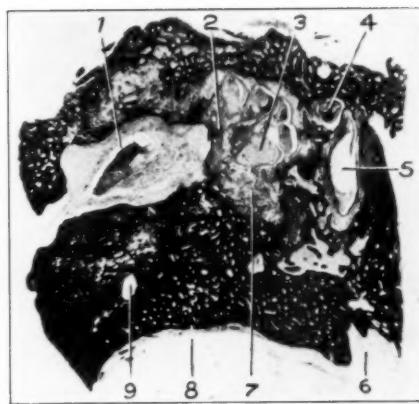
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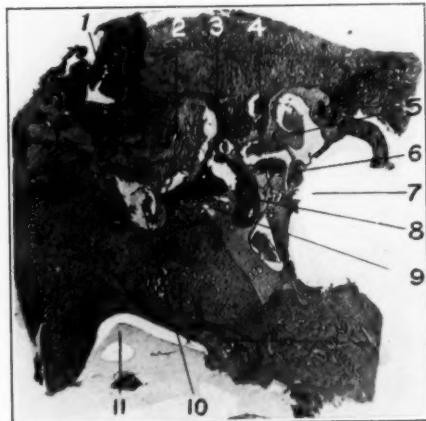
Fig. 67.

Fig. 68.

Fig. 69.—William Newberry. Vertical transverse section through oval window. 1, Facial nerve; 2, footplate of stapes displaced into vestibule, probably by contraction of fibrous tissue; 3, promontory; 4, round window membrane; 5, connective tissue in vestibule.

Fig. 70.—William Newberry. 1, New bone formation in vestibule; 2, new connective tissue formation; 3, abscess cavity; 4, niche of round window; 5, eroded promontory; 6, oval window; 7, facial nerve.

Fig. 71.—William Armstrong, aged 5. Chronic middle ear suppuration, right ear; complete deafness; latent labyrinthitis passing on to spontaneous cure; death from meningitis after operation. Horizontal section through inner ear. 1, Remains of abscess in cochlea; 2, vestibule filled up by new bone; 3, posterior canal filled up by new bone; 4, aqueduct of vestibule; 5, internal meatus showing nerves surrounded by meningitis; 6, new bone formation in cochlea; 7, normal cartilage bone capsule of cochlea; 8, normal lamellar bone.

Fig. 72.—Margaret Wood, aged 52. Chronic middle ear suppuration, right ear; spontaneous cure of labyrinthitis; thrombosis of sigmoid sinus and jugular vein; death from pyemia. Horizontal section through cochlea. The figure shows—1, that the whole of the cochlea has become a solid mass of bone; 2, internal meatus containing markedly atrophied nerves; 3, dilated saccule.

Fig. 69.

Fig. 70.
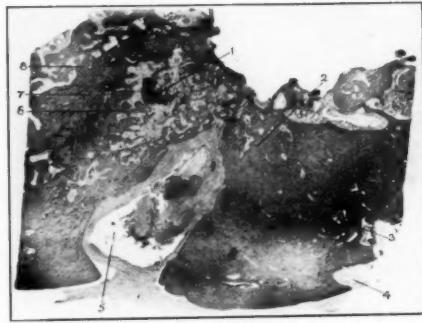
Fig. 71.

Fig. 72.

Fig. 73.—Margaret Wood. Horizontal section through oval window. 1, Facial nerve; 2, cystic space containing exudate; 3, footplate of stapes; 4, dilated saccule; 5, dilated utricle.

Fig. 74.—Case 1. W. Young, aged 1¼ years. Vertical transverse section of right ear, showing tubercular infiltration and erosion of walls of tympanic cavity. 1, Basal coil of cochlea; 2, tubal portion of tympanic cavity; 3, tensor tympani; 4, tubercular tissue in roof of cavity; 5, cochlear nerve in internal auditory meatus.

Fig. 75.—Case 1. Vertical transverse section, showing invasion of vestibule through oval window. 1, Membrane of round window; 2, tubercular erosion of promontory; 3, head of stapes; 4, tubercular tissue in niche of oval window; 5, facial nerve; 6, vestibular nerve to utricle, external and superior canals; 7, footplate of stapes eroded and displaced towards vestibule; 8, vestibular nerve to ampulla of posterior canal; 9, tubercular tissue filling up niche of round window.

Fig. 76.—Case 1. Vertical transverse section through canals. 1, Two ends of lateral canal; 2, fossa subarcuata infiltrated with tubercle; 3, two ends of posterior canal; 4, stapedius; 5, facial nerve; 6, erosion of bone over lateral canal prominence.

Fig. 73.
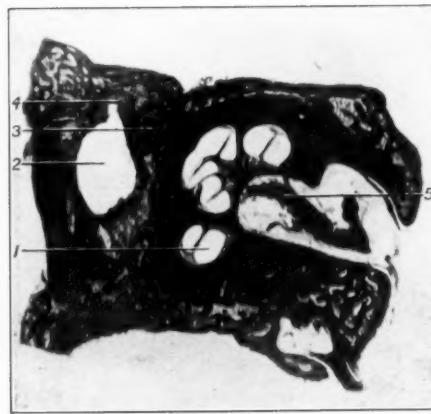
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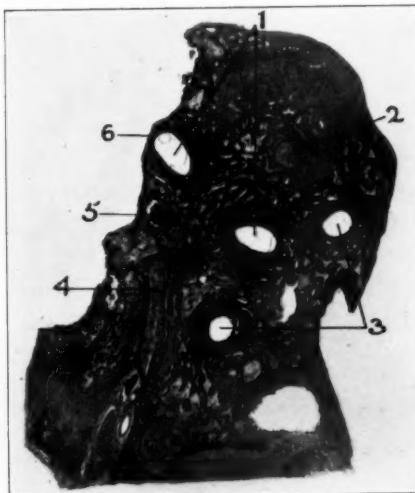
Fig. 76.

Fig. 77.—Case 2. J. Nisbet. Advanced tubercular disease of the ear. Vertical transverse section through apex of petrous bone. 1, Region of eustachian tube. The cartilaginous tube has disappeared. Caseating tubercle is seen to the left of and below the internal carotid artery. 2, Internal carotid; 3, apex of petrous bone; in this region the bone consists largely of marrow spaces.

Fig. 78.—Case 2. Vertical transverse section through anterior part of petrous pyramid. 1, Internal carotid artery; 2, tubercular granulation tissue infiltrating the wall of the carotid artery; 3, region of eustachian tube; the tube can no longer be recognized; 4, anterior part of petrous pyramid.

Fig. 79.—Case 2. Vertical transverse section through cochlea. 1, Fistula into basal coil of cochlea; 2, middle coil of cochlea—all scalae are filled with tubercular granulation tissue; the osseous spiral lamina is still to be seen; 3, fistula into apex of cochlea; 4, cartilage bone capsule of cochlea; 5, lamellar bone surrounding cochlear capsule; 6, tubercular pachymeningitis in floor of middle cranial fossa; 7, facial nerve; 8, auditory nerve; 9, cellular infiltration in fundus of internal meatus.

Fig. 80.—Case 2. Vertical transverse section through semicircular canals. 1, Erosion of bone by tubercular granulation tissue; 2, ampullary end of superior canal; 3, tubercular granulation tissue in fossa subarcuata; 4, smooth end of superior canal (perilymph space); 5, endolymphatic space of smooth canal; 6, saccus endolymphaticus (normal); 7, smooth end of posterior semicircular canal with granulation tissue.
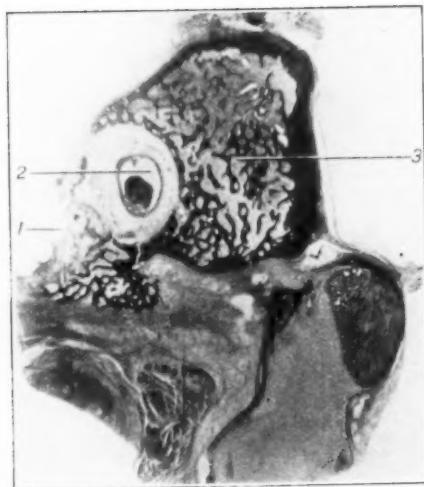
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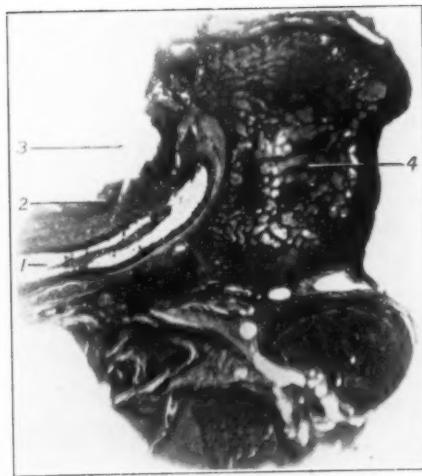
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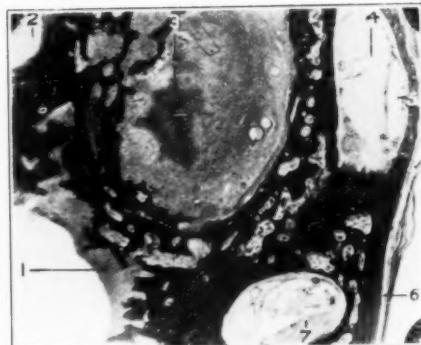
Fig. 80.

Fig. 81.—Case 3. John Cunningham. Vertical section through cochlea. 1, Facial nerve; 2, dilated scala media of middle coil; 3, fistula between scala vestibuli of basal coil and tympanum; 4, basal coil of cochlea filled up by new connective tissue and bone; 5, internal meatus with meningitic infiltration; 6, new formed bone filling up scala vestibuli.

Fig. 82.—Case 3. Section through middle coil of cochlea. 1, Bony spiral lamina; 2, Reissner's membrane pulled inwards by contraction of connective tissue; 3, dilated cochlear canal; 4, small celled infiltration; 5, basilar membrane; 6, remains of Corti's organ; 7, scala tympani filled by connective tissue.

Fig. 83.—Case 3. 1, New connective tissue and bone in wall of vestibule; 2, middle cranial fossa; 3, facial nerve; 4, dehiscence in facial canal; 5, large fistula into vestibule in region of oval window; the promontory has disappeared; 6, tubercular sequestrum in lower part of vestibule.

Fig. 84.—Case 3. 1, New formed bone partially filling up vestibule and region of ampullary end of posterior canal; 2, middle cranial fossa; 3, dehiscence in facial canal—caused by tubercular erosion (?); 4, tubercular granulation tissue in center of vestibule.
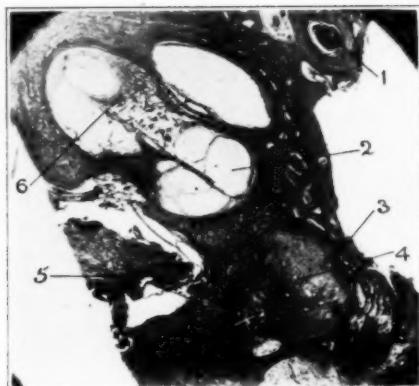
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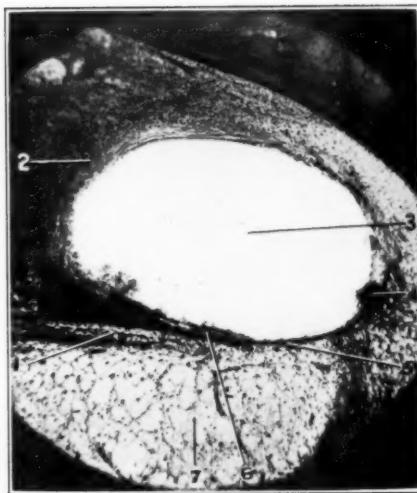
Fig. 82.

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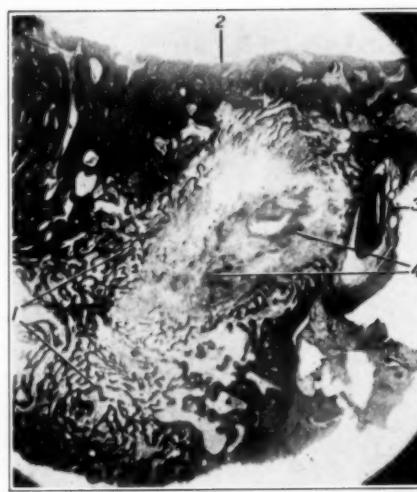
Fig. 84.

Fig. 85.—A case with Meniere's symptoms and facial paralysis. Vertical section of cochlea and internal auditory meatus. 1, Small cell infiltration in internal meatus; 2, above the seventh nerve; 3, beneath the epineurium.

Fig. 86.—Case 1. Tumor of right nervus acusticus. Horizontal section of right ear. Axial section through cochlea and internal meatus. 1, Scala media of upper part of middle coil, showing Corti's organ; 2, new bone formation in scala tympani; 3, saccule; 4, new connective or tumor tissue in scala tympani of basal coil; 5, erosion of cartilage capsule of cochlea by tumor tissue; 6, cochlear canal of lower part of basal coil, showing absence of Corti's organ and degeneration of spiral ligament.

Fig. 87.—Case 1. Horizontal section through basal coil of cochlea (right ear). 1, Reissner's membrane; 2, spiral canal with marked atrophy of ganglion cells; infiltration by delicate connective tissue; 3, delicate connective or tumor tissue in scala tympani; 4, large vein; 5, new formed bone; 6, myxomatous or dropsical degeneration of spiral ligament; 7, stria vascularis—note absence of Corti's organ.

Fig. 88.—Case 1. Horizontal section of right ear. 1, Tympanic membrane; 2, serous exudate in niche of round window; 3, connective or tumor tissue in scala tympani of basal coil; 4, cochlear nerve in fundus of internal meatus; 5, site of tumor which has been removed; in the right hand bottom corner a piece of the tumor tissue still remains; 6, aqueduct of cochlea and accompanying vein; 7, two ends of posterior canal (note that the perilymph space at the smooth end is almost filled by connective tissue); 8, serous exudate in air cells.

Fig. 85.

Fig. 86.
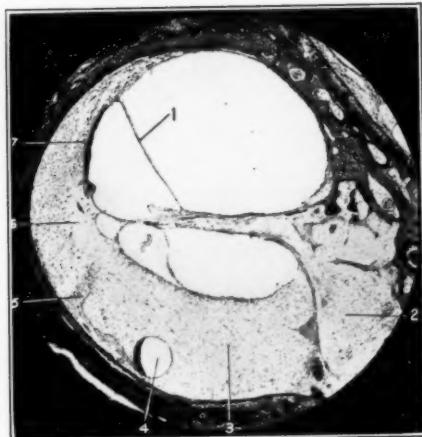
Fig. 87.

Fig. 88.

Fig. 89.—Case 1. Horizontal section of left ear. New connective tissue in scala tympani of apical coil; 2, greatly dilated cochlear canal of upper part of middle coil; note new connective tissue and bone in scala vestibuli; 3, spiral ganglion cells still present; 4, cochlear nerve; 5, hemorrhage between dura mater and bone; 6, scala tympani of lower part of basal coil with new connective tissue and bone formation; 7, scala vestibuli of middle coil filled with new bone formation.

Fig. 90.—Case 2. Tumor of eighth nerve. Horizontal section of right ear. 1, Upper part of basal coil of cochlea; 2, facial nerve—the tumor tissue is infiltrating as far as the geniculate ganglion; 3, tumor infiltrating vestibular nerve to utricle; 4, utricle; 5, facial nerve; 6, convexity of lateral canal; 7, smooth end of posterior canal; 8, erosion of bone by tumor; 9, crus commune; 10, tumor.

Fig. 91.—Case 2. Horizontal section of right ear. 1, Head of stapes with stapedius; 2, facial nerve; 3, posterior canal with hemorrhage in perilymph space; 4, tumor; 5, lower part of utricle; 6, dilated internal meatus with tumor.

Fig. 92.—Case 2. Horizontal section of right ear. 1, Thickened membrane of round window; 2, new bone formation in connective or tumor tissue in scala tympani; 3, spiral canal infiltrated and ganglion cells absent; 4, remains of cochlear nerve in fundus of internal meatus; 5, canal for nerve to crista of posterior canal infiltrated as above; 6, endolymph space of posterior canal.
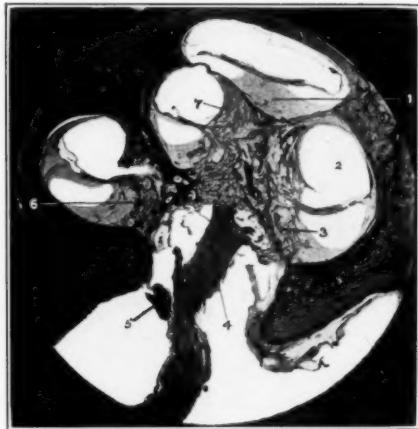
Fig. 89.

Fig. 90.

Fig. 91.
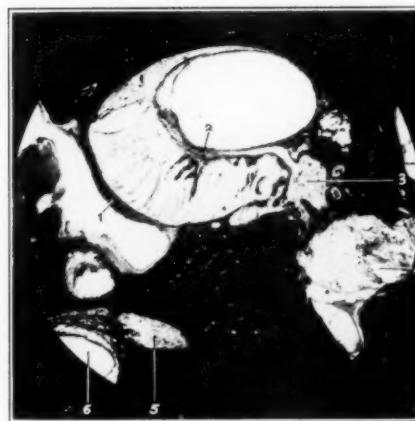
Fig. 92.

Fig. 93.—Case 2. Shows tumor of the pia arachnoid, which occupied one of the small bony erosions. The section shows a fibrous, cellular growth, apparently composed of neuroglial tissue. The neuroglial fibers appear to run in bundles in various directions. Cushing holds that these swellings are hypertrophied Pacchionian bodies, while Key believes that they are gliomatous metastases in the arachnoid. They certainly appear to be of a gliomatous nature.

Fig. 94.—Case 3. Horizontal section of right ear. 1, External meatus; 2, malleus; 3, tensor tympani; 4, gasserian ganglion; 5, tumor; 6, dilated saccule with granular contents; 7, utricle; 8, smooth end of posterior canal; 9, lateral canal; 10, footplate of stapes; 11, long process of incus.

Fig. 95.—Case 3. Axial section through right cochlea. 1, Dilated cochlear canal, basal coil; 2, capsule of tumor; 3, tumor tissue; 4, cochlear nerve compressed by tumor tissue; 5, central canal of modiolus.

Fig. 96.—Case 3. Axial section through right cochlea. 1, Fibrinous and granular exudate in scala vestibuli; 2, dilated cochlear canal; 3, stria vascularis; 4, fibrin threads in scala tympani; 5, spiral canal; some ganglion cells are still to be seen; 6, nerve entering bony spiral lamina.

Fig. 93.

Fig. 94.

Fig. 95.

Fig. 96.
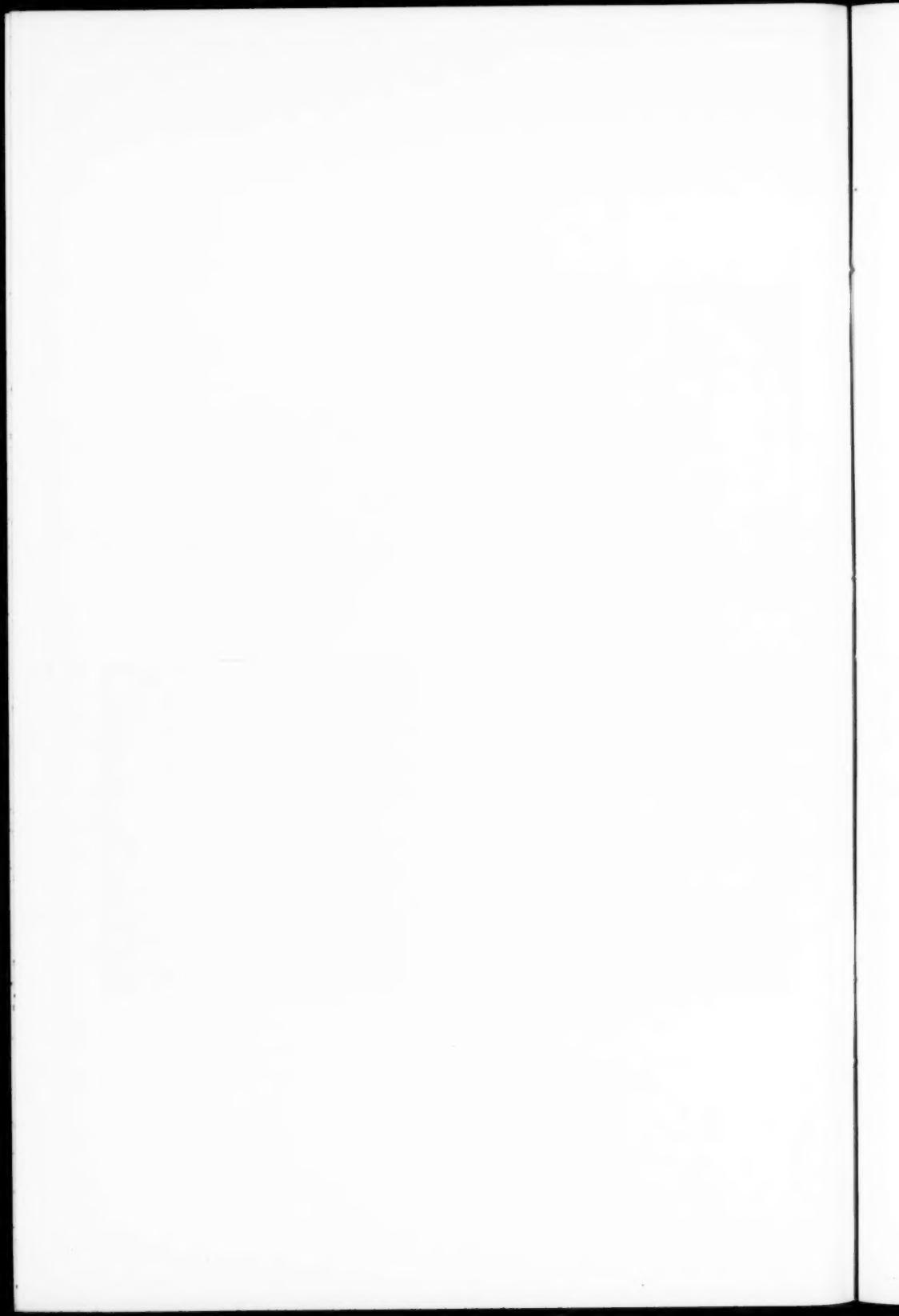
Fig. 97.

Fig. 97.—Case 3. Horizontal section of right ear. 1, External meatus; 2, hemorrhage in tubal portion of tympanic cavity; 3, internal carotid artery; 4, tumor; 5, cochlear opening of perilymphatic aqueduct; 6, region of saccus; 7, two parts of posterior canal.

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LXXI.

OBSERVATIONS OF ADENOIDS REMOVED WITH A DIRECT VISION ADENOTOME.

By I. D. KELLEY, JR., M. D.,

ST. LOUIS.

Since Wilhelm Meyer[1] directed the profession's attention to adenoids, various instruments for their removal have been devised. The most popular in use are perhaps the Gottstein, Beckmann and Hartmann curets, the adenotomes or nasopharyngeal tonsillotomes of La Force and Schutz-Passow, and the Jurasz adenoid biting forceps. Each of these instruments employs a blind technic in adenoid removal.

From time to time, efforts have been made to remove adenoids under direct vision, sometimes by means of a palate retractor held in the left hand while the operation is done with the right hand. Recently, Joseph C. Beck[2] has employed rubber catheters passed through the nostrils and out of the mouth, which are tied, serving in this way as an elastic palate retractor. None of these efforts in my experience have been so satisfactory as the removal by the direct vision adenotome. Observation on the cadaver showed that a straight line was possible from the eye of the observer to the upper limit of the adenoid with this instrument in position. This is easily demonstrable on the living subject and has been the incentive in the development of this instrument and its use.

Figures 1 and 2 show the direct vision adenotome. The instrument consists of a hollow shaft or hood, which permits direct vision and serves as a soft palate retractor, the distal or blade end of which, when put into position, conforms to and rests directly against the posterior nasopharyngeal wall. From the proximal end of the shank extends a hand grip, placed at an angle that will prevent chin or chest interference

when gripped and placed in position. On the upper surface of the shank is a shaft, to which is attached a flexible blade traversing the cutting surface at the distal end of the hood from below upward, thus permitting adenectomy under the vision of the surgeon.

The blind technic previously employed in adenoid removal is responsible for much of the diversity of opinion regarding adenoids found among operators in our clinics, which the routine use of the direct vision adenotome will help to make clear. When adenoids are removed with this instrument, the nasopharynx is encircled sufficiently by the cutting surface to show the adenoids entirely surrounded and presenting a definitely organized appearance, easily differentiated from the surrounding mucous membrane. The instrument can be so exactly placed about the adenoid, and such definite pressure exerted on the soft structure of the posterior wall, that the blade, when it engages the adenoid, is forced behind, severing it with its capsule. This exactness in technic, which is due to direct vision, has proved so unfailing that a large number of adenoid cases, checked by a careful and searching digital examination of the nasopharynx after operation, have shown the postnasal space entirely freed from lymphoid tissue. A removed adenoid has been likened, because of its supposedly loose construction, to the leaves of a poorly bound book spread apart and suspended, causing the leaves immediately to fall and scatter. As a matter of fact, my experience has been that adenoids removed with such precision as this instrument affords are quite well organized, owing, no doubt, to the presence on their cut surface of a definite fibrous capsule.

This ability to bring the adenoid into complete vision, coupled with the constant finding of a definitely organized mass of lymphoid tissue, indicates clinically that adenoids have a definite location in the nasopharynx. When we find by digital examination what seems to be a wild proliferation of adenoid tissue filling the postnasal space, it is by mass extension of the lymphoid units composing the adenoid and not the actual spread or proliferation of its basal attachment beyond easily recognized limits.

Barnes[3] states, in his description of the location of adenoids, that the larger central adenoid mass occupies the median portion of the posterior wall with scattered lymphoid masses, extending into Rosenmüller's fossa, often filling it. I believe that if we embrace all lymphoid tissue found in this region under the term "adenoids," we should not only include the lymphoid tissue in Rosenmüller's fossa, which is an extension upward of that part of Waldeyer's ring known as the posterior lymph column, but must include as well the small isolated lymph patches on the posterior wall. This term cannot embrace all these structures, because in adenoids removed with direct vision we are dealing with a definitely located encapsulated mass of lymphoid tissue, which I feel is synonymous with the nasopharyngeal tonsil, and which is shown to have its attachment extending above to the posterior superior border of the nasal septum, below to the belly of the superior constrictor muscle, and laterally to the posterior lymph columns, found in hypertrophied adenoids as compared to the smaller atrophic organized adenoids occupying a lesser portion of this area.

Figure 3 represents two views of an adenoid which was completely surrounded and removed in direct vision. On the anterior or presenting surface are seen the several units composing the adenoid, represented by the crypts surrounded by eminences of lymphoid tissue. On the posterior or capsular surface is seen a well organized fibrous capsule with nodules and depressions similar to those found on the tonsil. Figures 4 and 5 are sections taken at random from many adenoids removed with their capsules, and show their histologic structure under low magnification. In the lowest magnification is seen the same histologic structure as the tonsil. At the top is a well developed capsule from the tunica propria of the mucous membrane with definite fibrous trabeculæ descending into the lymphoid mass, composed of reticular connective tissue containing countless leukocytes in its meshes, and scattered throughout are the germinal centers or oval rings of lymphocytes. The higher magnification shows more clearly the tunica propria or capsule on which the adenoid is developed, its numerous mucous glands and blood vessels, together with the

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trabeculæ forming the coarser framework of the lymphoid tissue. The capsular structure is seen to be so well marked that it must serve to show that the previous methods of adenoid removal were responsible for much of the seeming lack of organization found in this tissue.

The direct vision adenotome permits a study of the adenoid immediately before and during removal. Many interesting forms of adenoid inflammation are seen, from simple blocking of the crypts with débris to suppurative follicular adenoiditis.

Figure 6 represents one of several adenoids removed where pus was seen to exude from a small opening on the adenoid surface; and while the adenoid was being severed from its attachment a long, wormlike mass of débris exuded from the opening of the pharyngeal bursa, partially filling the hood of the adenotome. In the left view is seen the presenting or anterior adenoid surface with a bristle inserted into the opening; while to the right is the posterior surface with the capsule partially removed, revealing multiple abscesses occupying the body of the adenoid. Figure 7 is a section through the adenoid shown in Figure 6, above the level of the abscesses, showing the pus track occupying the centers of the largest adenoid mass; while Figure 8 is a section through the multiple abscesses occupying the body of the adenoid. In the section at the top is also seen a portion of the adenoid capsule with its trabeculæ descending toward the epithelial surface.

Thornwaldt[4] describes a disease of the adenoid in which pus is seen to exude from the median bursa that has partially closed, as the result of inflammation, and the symptoms ascribed to this disease are persistent and recurrent attacks of pharyngitis. The clinical findings in the case are shown in Figures 6, 7 and 8, at operation under direct vision; and because of the large quantity of pus expelled from the adenoid they would suggest much more than purulent infection of a partially closed bursa described in Thornwaldt's disease. This, together with the extensive involvement shown on microscopic examination, could no doubt produce the same symptoms caused by pus absorption from the tonsils, teeth or focal pus infection elsewhere in the body.

Figure 9 is inserted to show that the median adenoid bursa is not a necessary factor in the production of these suppurative adenoids. The left view of Figure 9 is the presenting surface of an adenoid with an opening containing a bristle to the side of the median bursa, from which at operation an unusually large quantity of pus was expressed. To the right is the capsular surface with the capsule seen to be removed intact, with a protruding eminence in which pus was seen through the glistening semitransparent membrane. Figure 10 is a section through the same adenoid, showing the extensive involvement of the tissue surrounding the cavity. The cavity is surrounded by a wide necrotic area in which much of the lymphoid structure is destroyed.

When no adenoids are present, it is possible with the direct vision adenotome to study the smooth, glistening, velvet-like mucous membrane of the superior postnasal wall in contrast to the mucous membrane below covering the superior constrictor muscle which forms, when the superior constrictor is contracted, a raised border representing the upper edge of the muscle extending horizontally across the posterior nasopharyngeal wall, with pendicular rugæ running into the pharynx. It is this upper raised border of the muscle which is cut into and the rugæ stripped during the curette operation when inspection reveals tags hanging in the throat after adenoid removal.

The technic of adenoid removal with the direct vision adenotome is as follows:

Under local or general anesthesia, the mouth is widely opened with a gag; the instrument is inserted in the mouth; the posetrior portion of the tongue is depressed with the under surface of the hood until above the upper surface is seen the free margin of the soft palate; the instrument is then gently pushed back to the posterior wall, and the distal or hood end is raised into the nasopharynx, the small ridge on the edge of the upper hood surface automatically engaging the soft palate border, forcing it forward on the surface of the hood out of harm's way. The distal end of the instrument is then further raised into the nasal vault until the adenoid is seen to be com-

pletely surrounded, when one looks through the hood, and in full view of the operator. The instrument is then pressed firmly against the posterior wall, and the razor sharp blade pushed closed by means of the thumb plate. The adenoid is thereby completely severed from its attachment, the operator seeing the blade cutting through the adenoid mass. When the instrument is removed, the adenoid will be found in a cup formed by the blade closing the inside of the hood.

Indications for the direct vision operation are found in all cases in which a complete surgical adenoid removal is desired. There are no contraindications to its use. Occasionally we find deep depressions on the posterior wall above the second cervical vertebra which the blade of this instrument, running as it does in a rigid arc, cannot reach; but this cannot be advanced as a contraindication, because even the blade of a curette used in the older operation is fixed between two rigid arms and, because of their interference, must traverse in its descent the same rigid arc. Therefore, to remove the remaining adenoid tissue in these depressions it is necessary to use a narrow curet, such as the one designed for such purposes by Sluder[5] (Figure 11), to reach these depressions, or biting forceps, which can best be done under direct vision through the hood of the direct vision adenotome. In several hundred observations we have found these depressions in only two cases. In cases in which the vault of the nasopharynx is unusually high, the use of the direct vision adenotome again has no contraindication if the technic of its introduction into the nasopharynx is properly carried out by feeling the definite resistance offered by the posterior superior border of the nasal septum and seeing the adenoid in complete vision.

When fragments of adenoid tissue are found not to have been removed from the vault, the resistance of the soft palate muscles is mistaken by the operator for the resistance felt when the upper distal border of the instrument comes in contact with the posterior superior border of the septum. Consequently, the vault has not been reached or the adenoid has not been completely surrounded and in full vision, as sometimes happens when the palate is not relaxed. Also, if the

operator releases his wrist tension when pushing the blade closed, the palate tension will force the cutting surface of the hood into a lower position on the posterior wall, thus permitting an upper unsevered portion of the adenoid to remain in the nasopharynx. With careful scrutiny of the adenoid in regard to complete vision during the cutting process, this error in technic should not occur.

Experience has shown in cases in which both tonsils and adenoids are to be removed that the direct vision adenotome should be used first, because the field of operation is unobscured. The "Sluder tonsillectomy" depends so little on the sense of sight, when properly understood, that a smear of blood in the pharynx is negligible. Therefore, removal of the adenoid should be the first operation done. However, in those unusual cases in which the tonsils are so large that the introduction of the adenotome into the nasopharynx is impossible, the tonsil should be first removed; the momentary gush of blood from the two operated fields is then allowed to stop, and the direct vision adenotome then introduced. Now, if the inside of the hood is sponged free of blood, full vision is permitted to complete the adenoid operation.

Frequently in clinics the use of the direct vision adenotome will reveal a wrong diagnosis. Cases are found in which the presence of adenoids is not noticed and the tonsils alone advised removed. Moreover, adenoids are often diagnosticated when their presence does not eixst. Operation under these conditions results in needless destruction of normal nasopharyngeal mucous membrane. Furthermore, with the routine diagnostic use of this instrument, it is surprising with what frequency adenoids are found in adults.

The use of the direct vision adenotome is advised as a diagnostic procedure in all tonsil and adenoid operations.

HUMBOLDT BUILDING.

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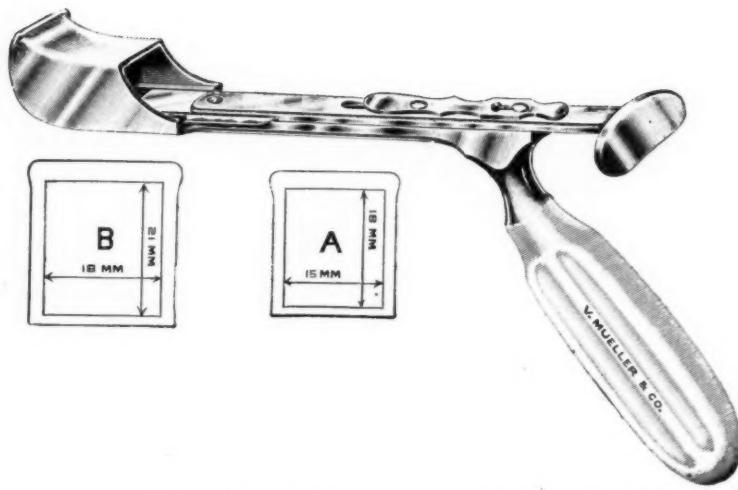
Fig. 1. Direct vision adenotome: A, dimensions of small instrument, to be used in children approximately under five years of age; B, dimensions of larger instrument, to be used in adults and children over 5 years of age.
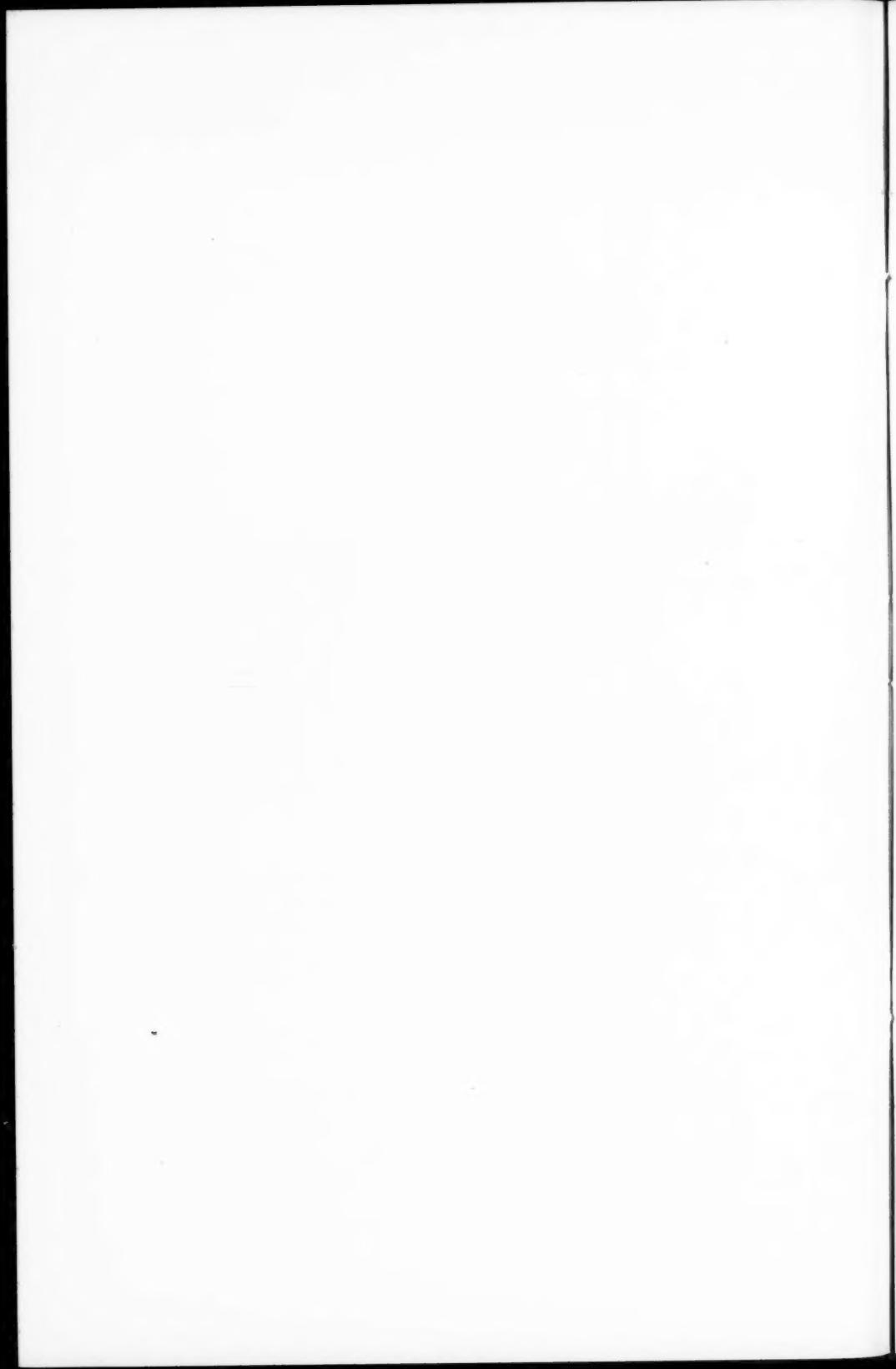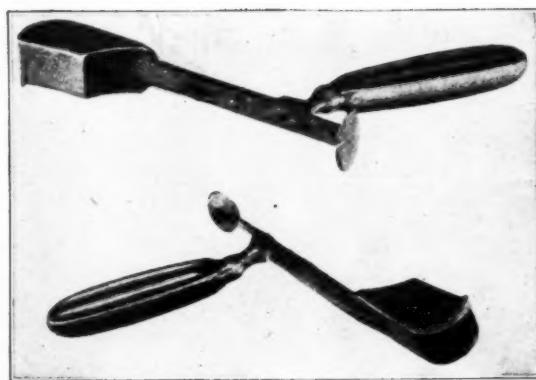
Fig. 2. Views of the direct vision adenotome.
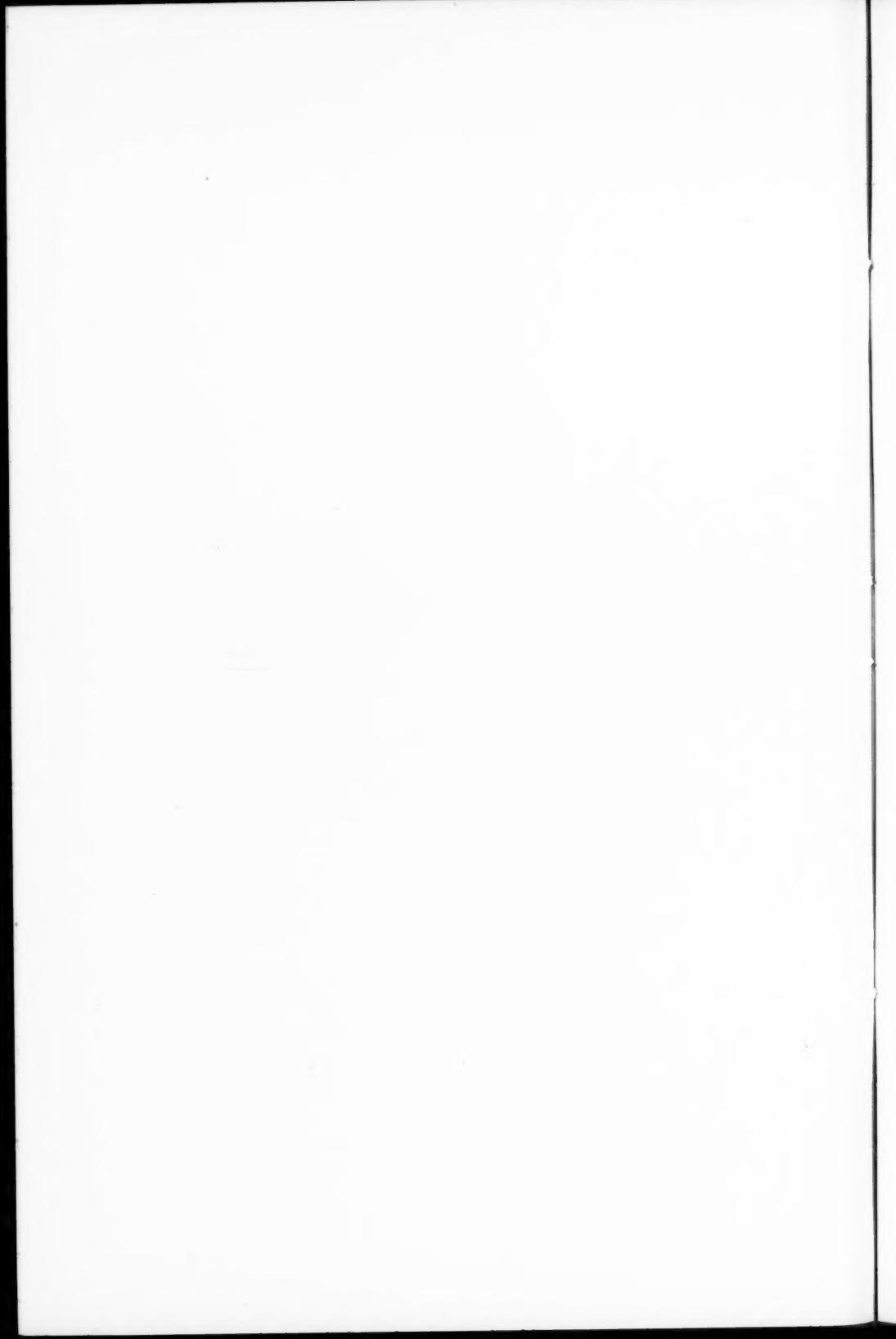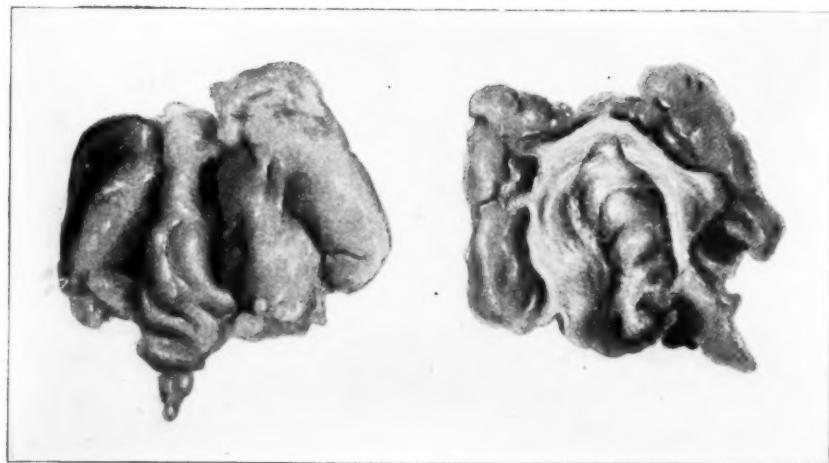
Fig. 3. Left view, anterior or presenting surface of an adenoid, showing crypts and elevations of lymphoid tissue; right view, posterior or capsular surface of adenoid, showing fibrous capsule with nodules and depressions similar to those found on capsular surface of the tonsil.
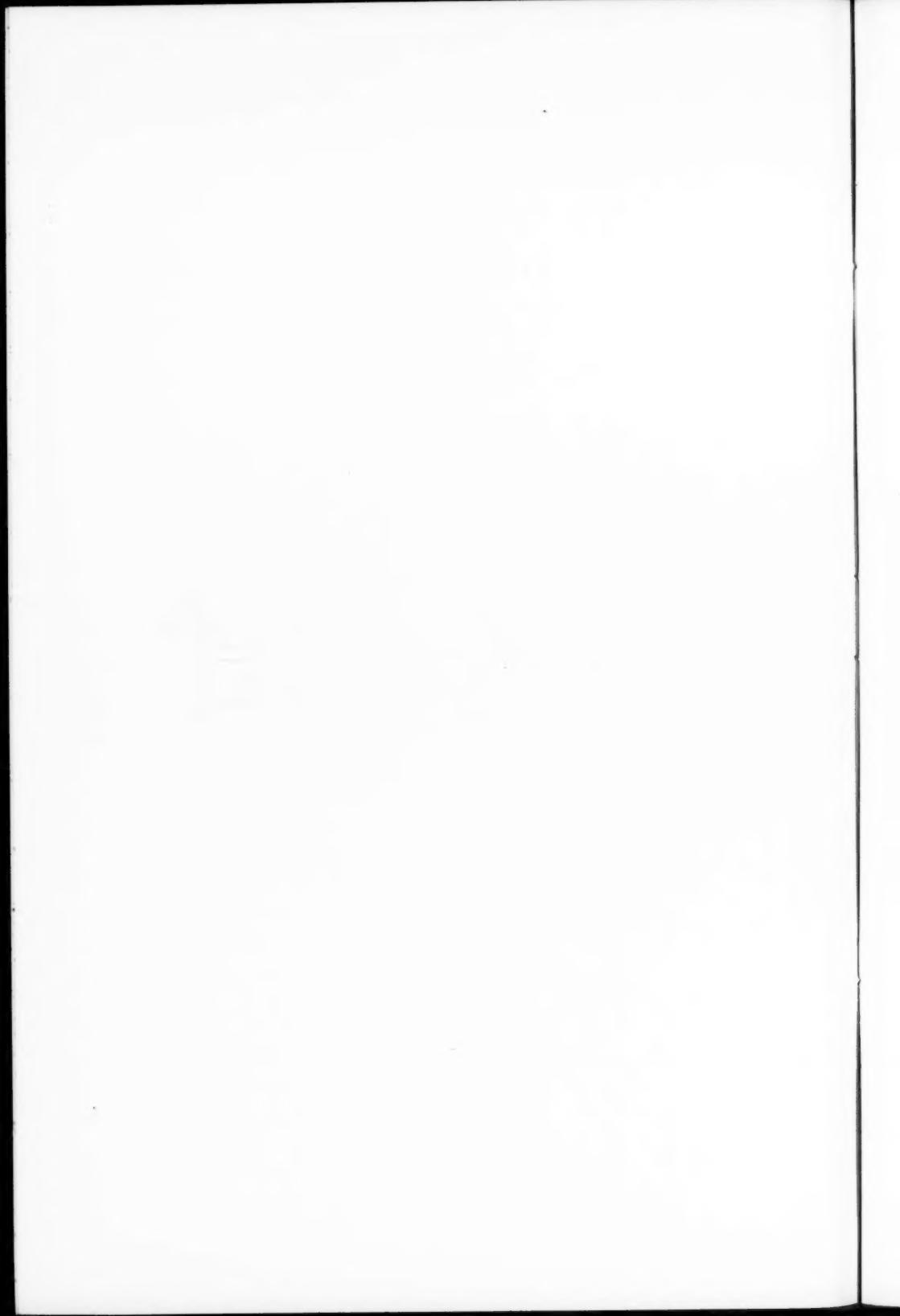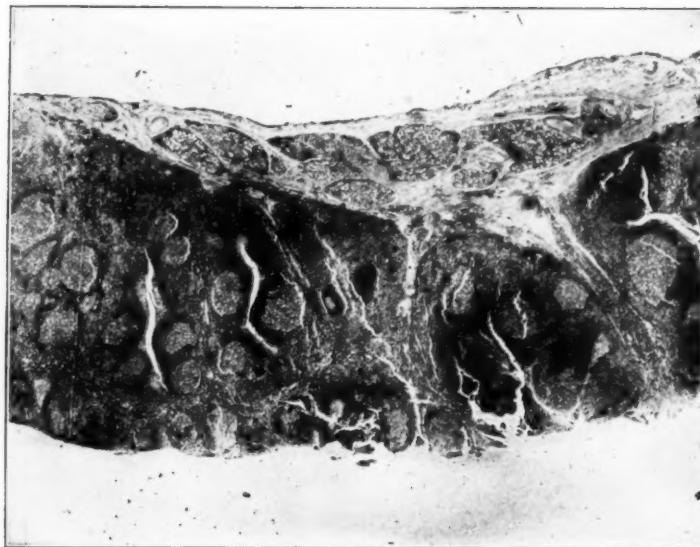
Fig. 4. Section through an adenoid, showing, above, a well developed capsule developed from the tunica propria of the mucous membrane containing mucous glands; fibrous trabeculae extend from the capsule toward the surface epithelium below; in the meshes of the reticular connective tissue are the leukocytes, among which are found numerous oval rings of lymphocytes or germinal centers.
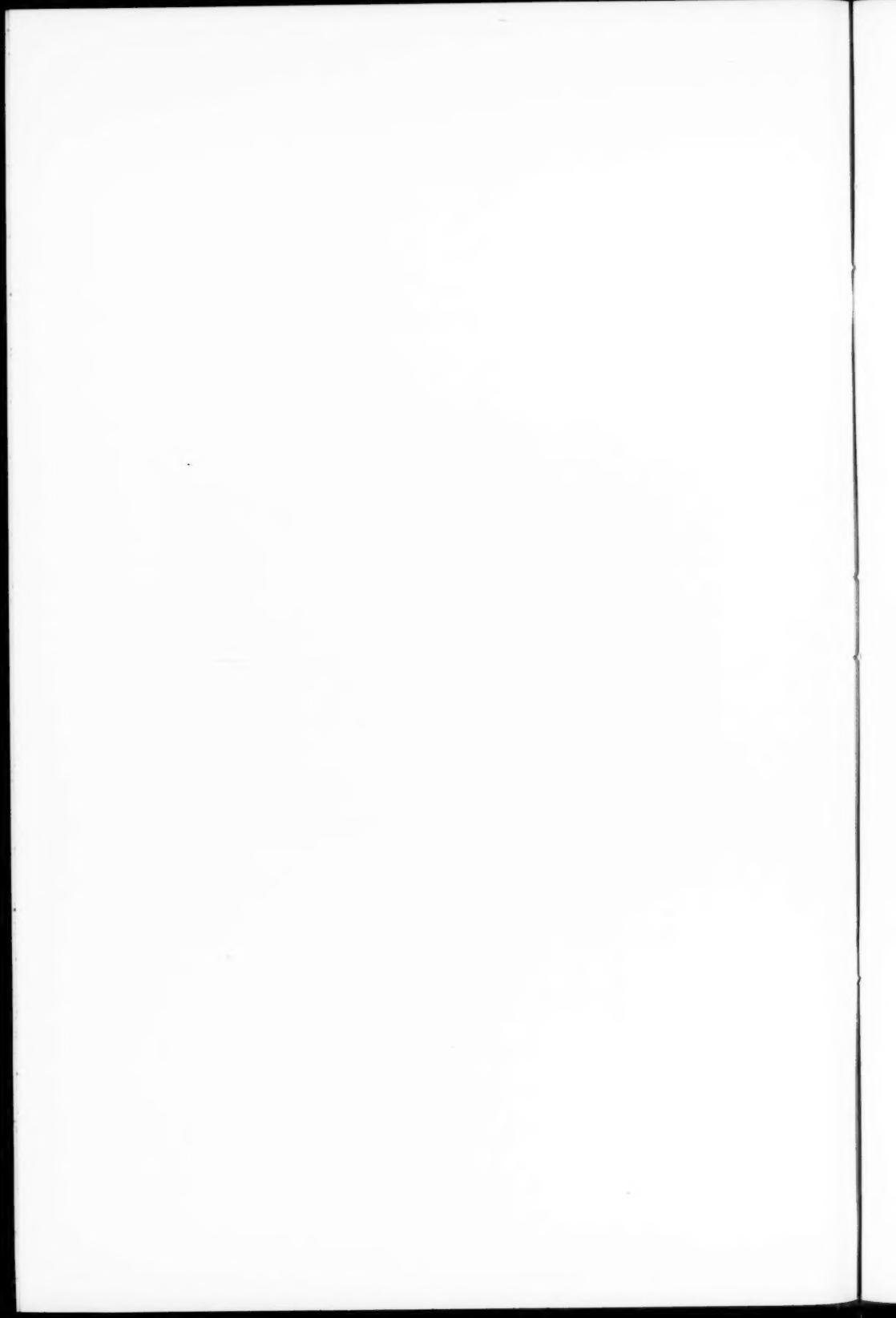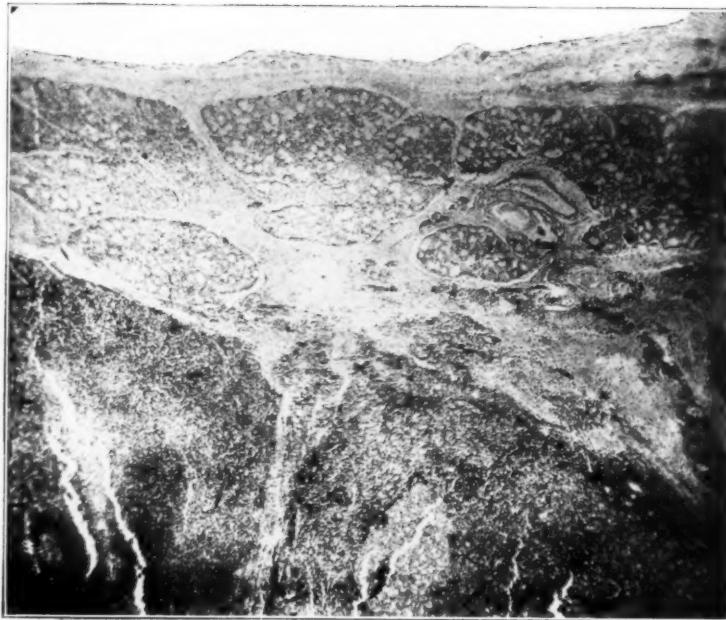
Fig. 5. Higher magnification of adenoid capsule, showing connective tissue of tunica propria, numerous mucous glands, and blood vessels and fibrous trabeculae from capsule descending into lymphoid tissue.
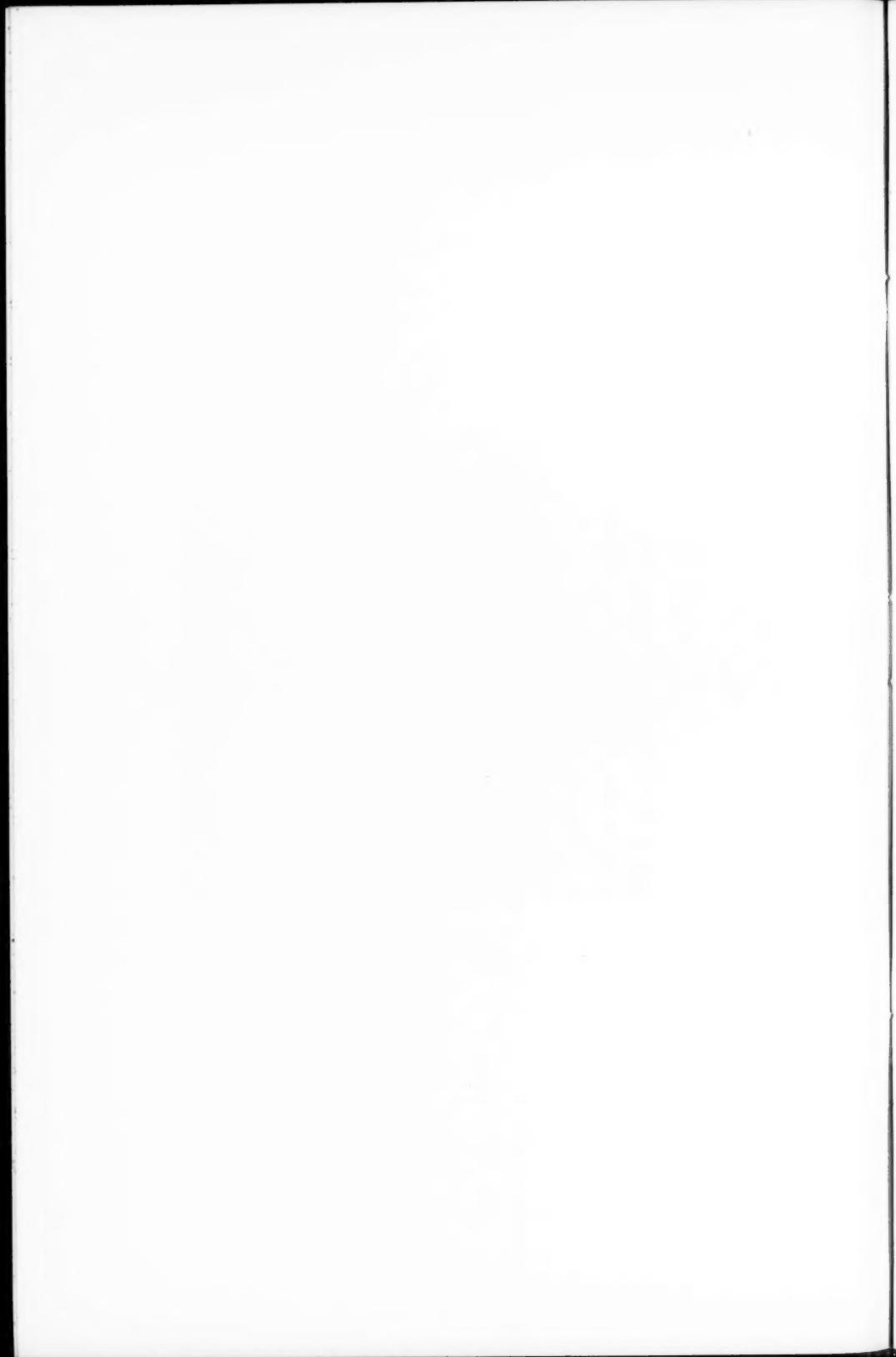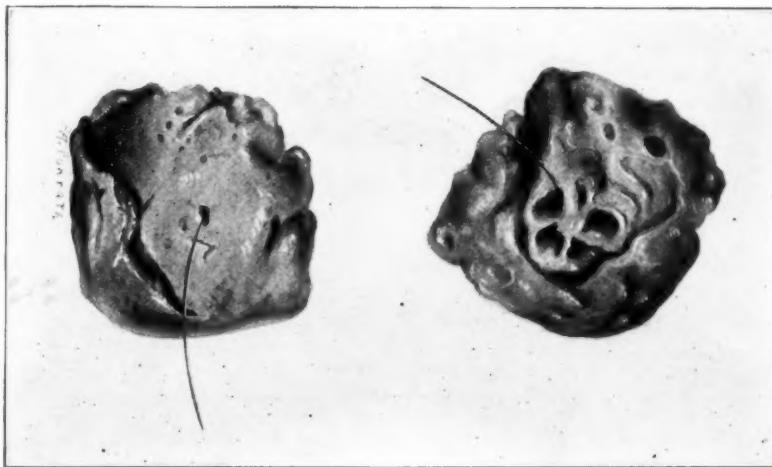
Fig. 6. Left view, anterior adenoid surface, showing opening with inserted bristle from which large quantities of pus and débris were seen to exude during removal; right view, posterior surface of adenoid, with capsule partially removed, showing multiple purulent abscesses occupying the body of the adenoid.
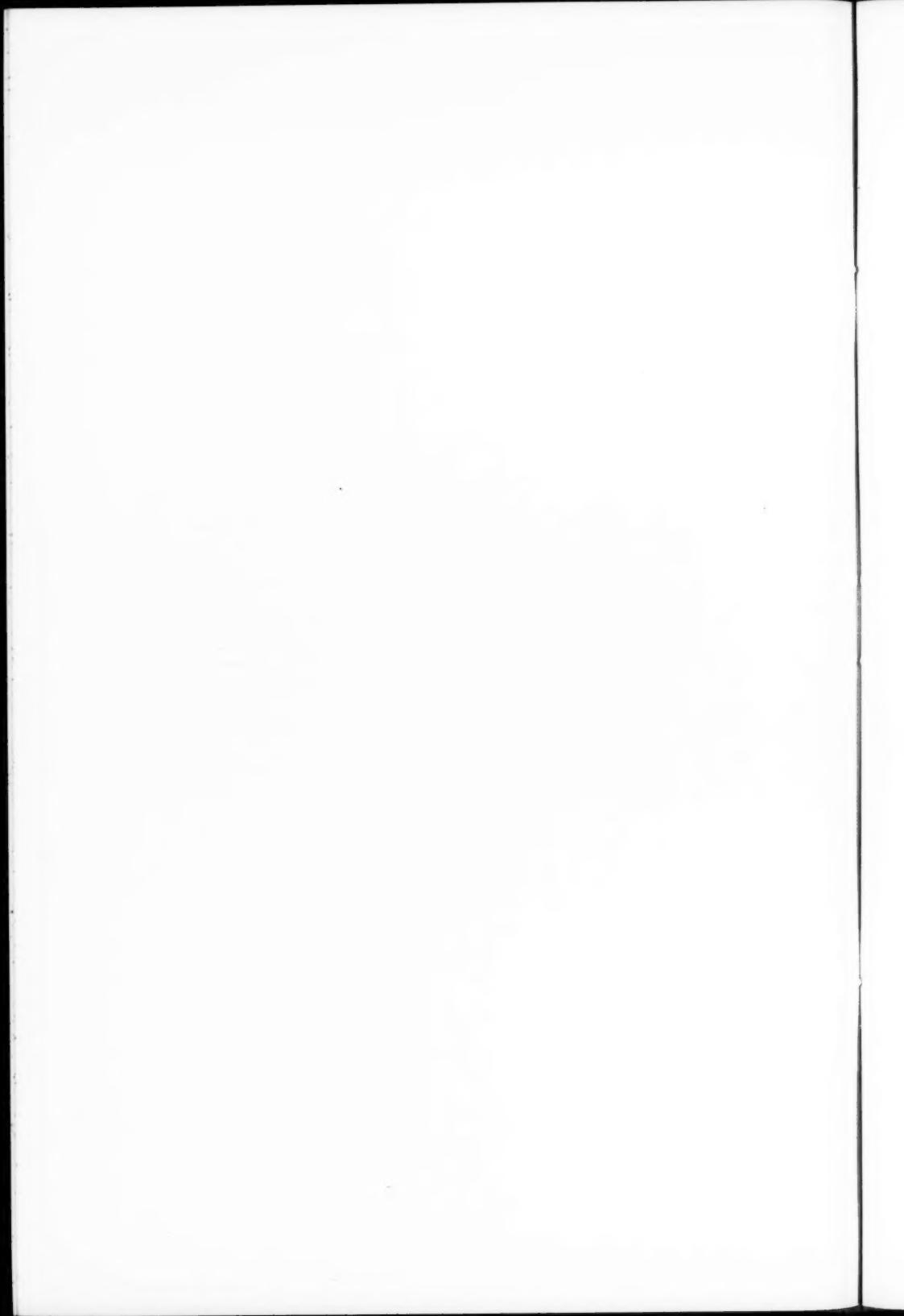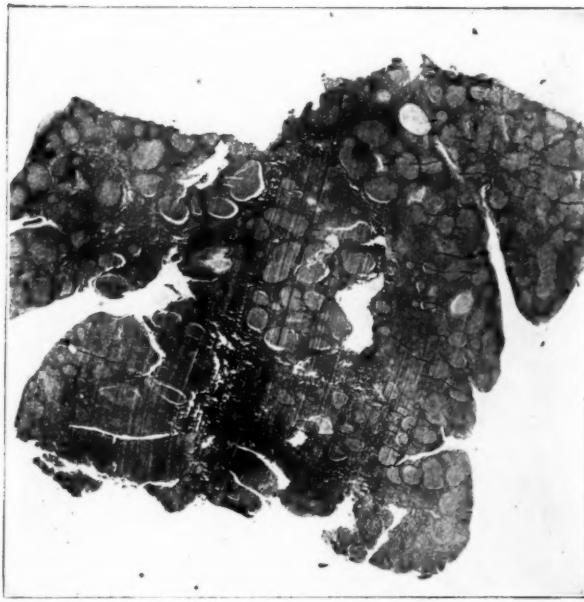
Fig. 7. Section through adenoid shown in Figure 6, with pus tract occupying center of largest adenoid mass.
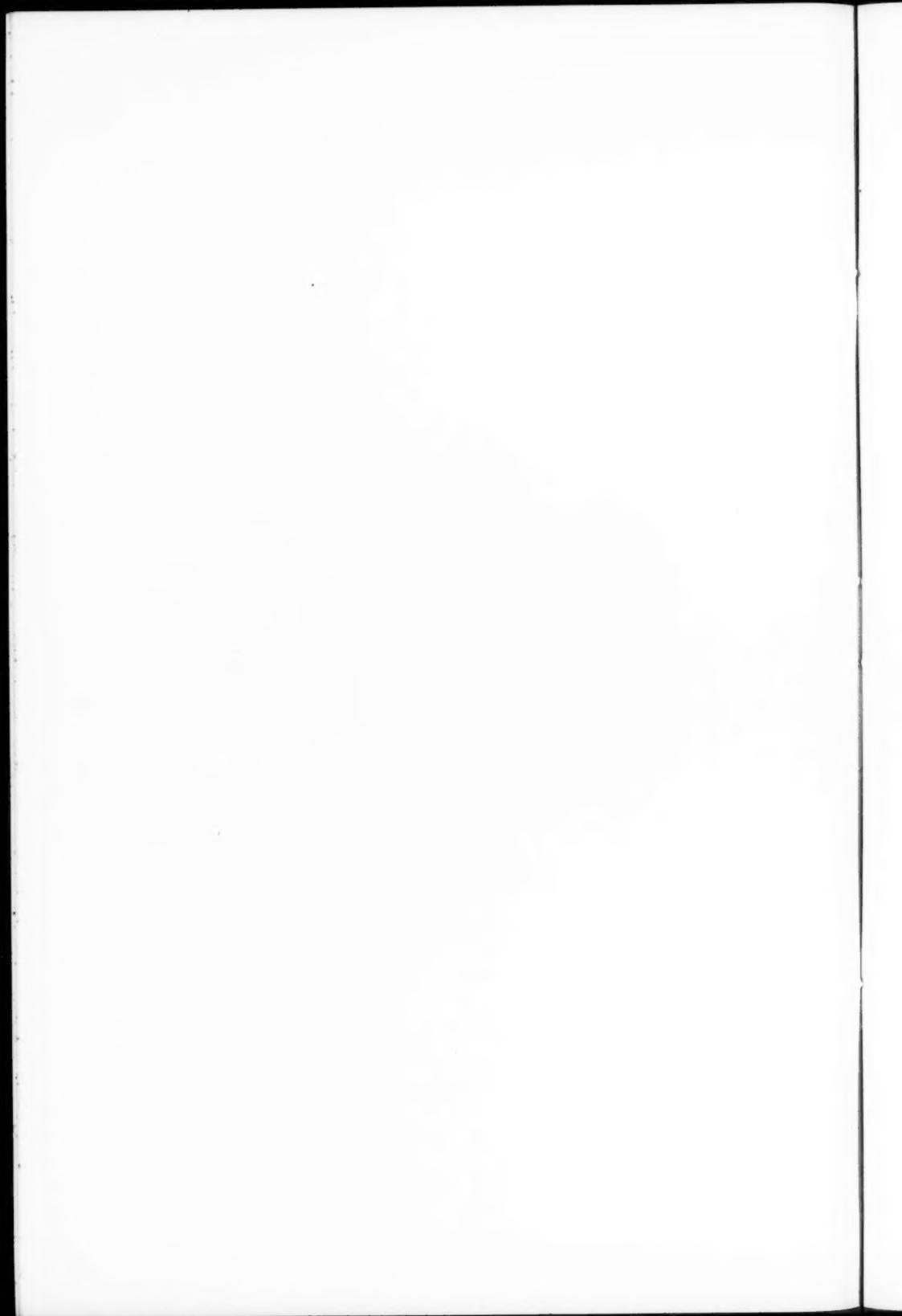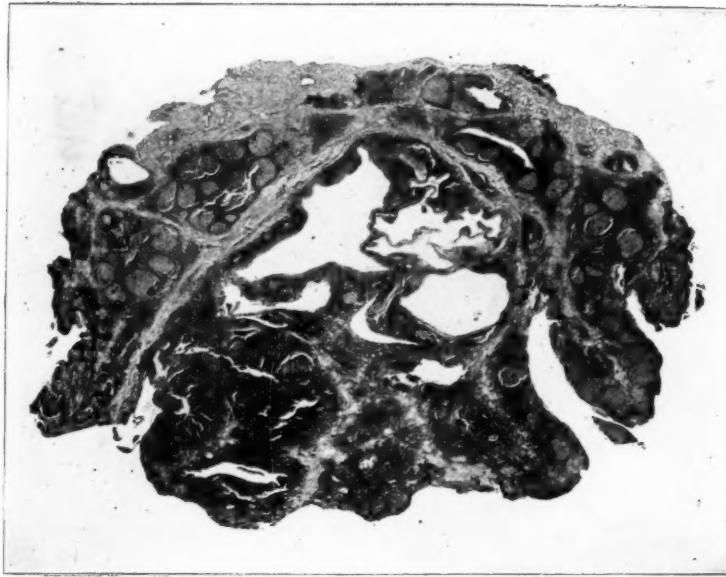
Fig. 8. Section through adenoid shown in Figure 7: Above is
seen the adenoid capsule with trabeculae descending toward the
epithelial surface, with the body of the adenoid occupied by large
abscesses.
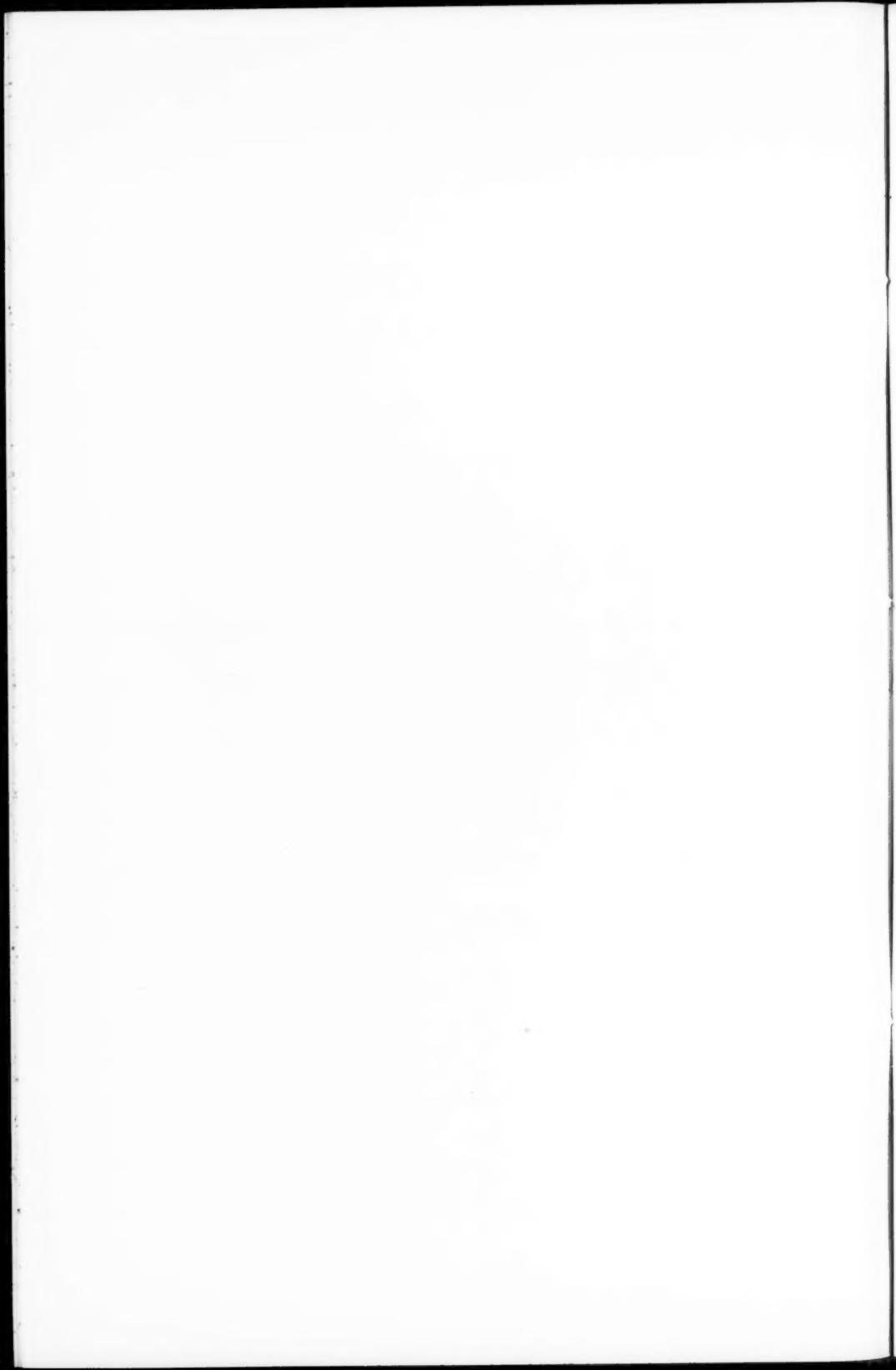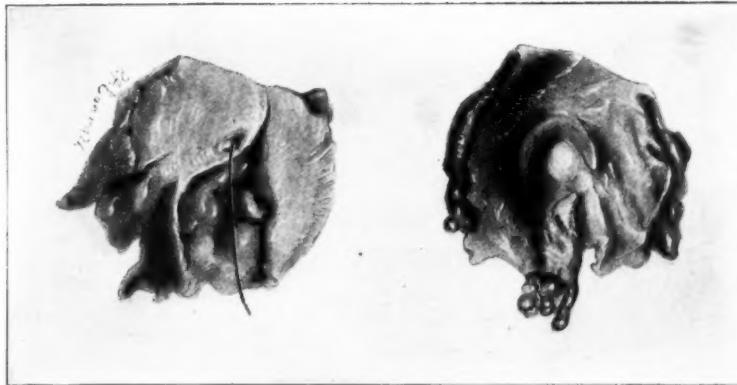
Fig. 9. Left view, opening containing a bristle on the presenting adenoid surface located to the side of the median bursa; right view, capsule of adenoid intact; the central elevated area was formed by a pus pocket seen through the capsule.
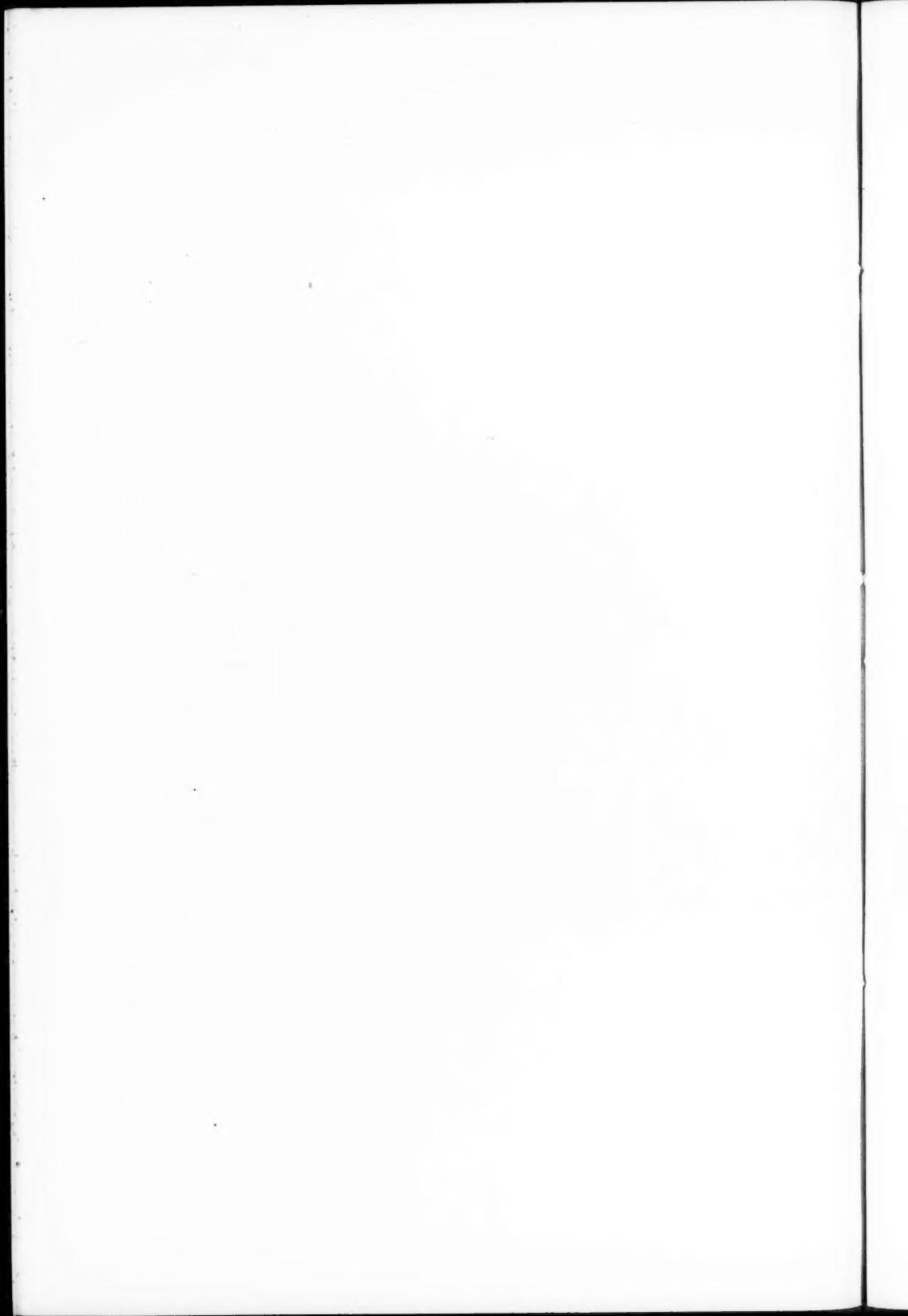
Fig. 10. Section from adenoid shown in Figure 9: A large necrotic area containing pus and destroying much of the lymphoid structure of the adenoid.
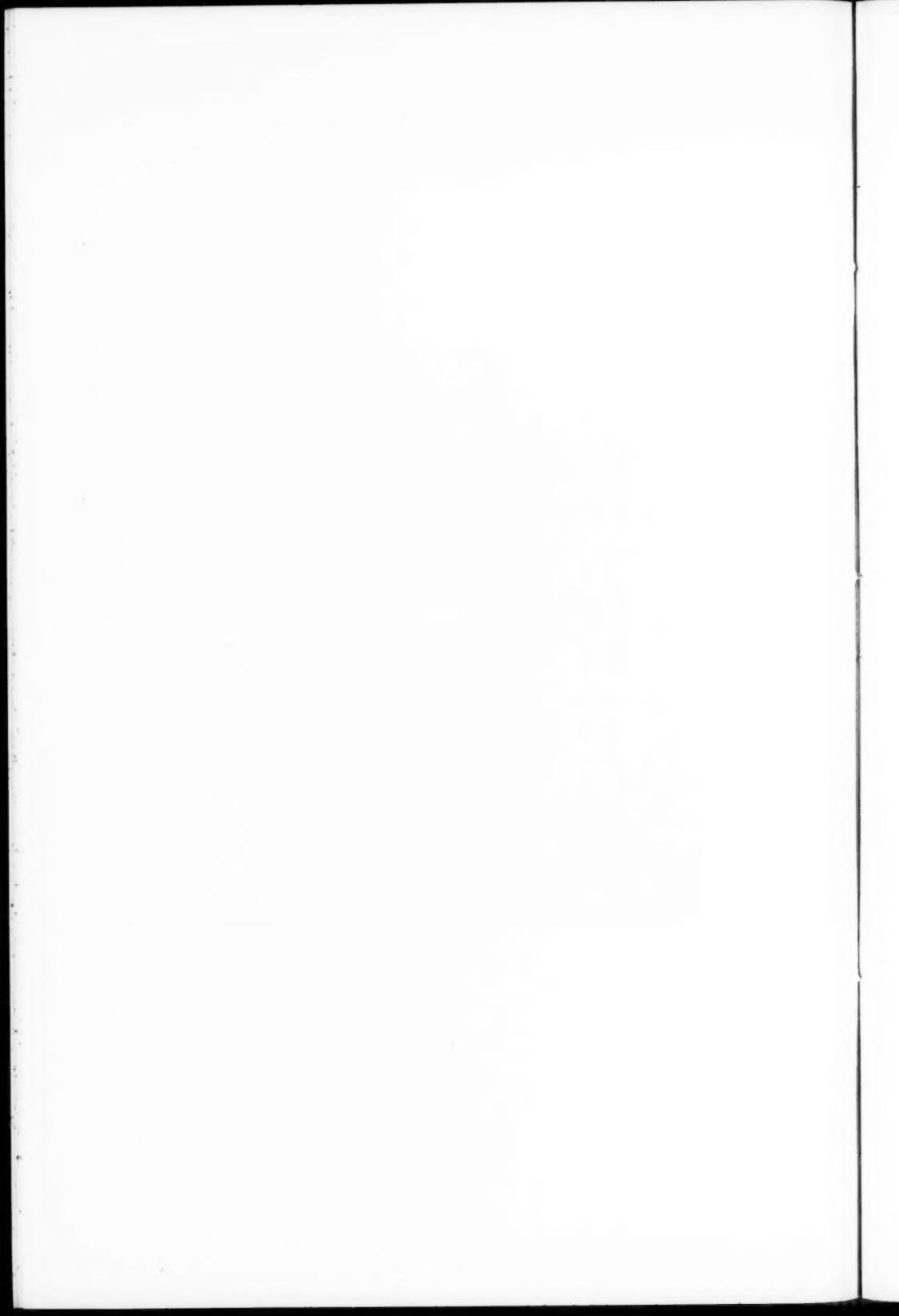
Fig. 11. Sluder's specially designed adenoid curet: A, closed;
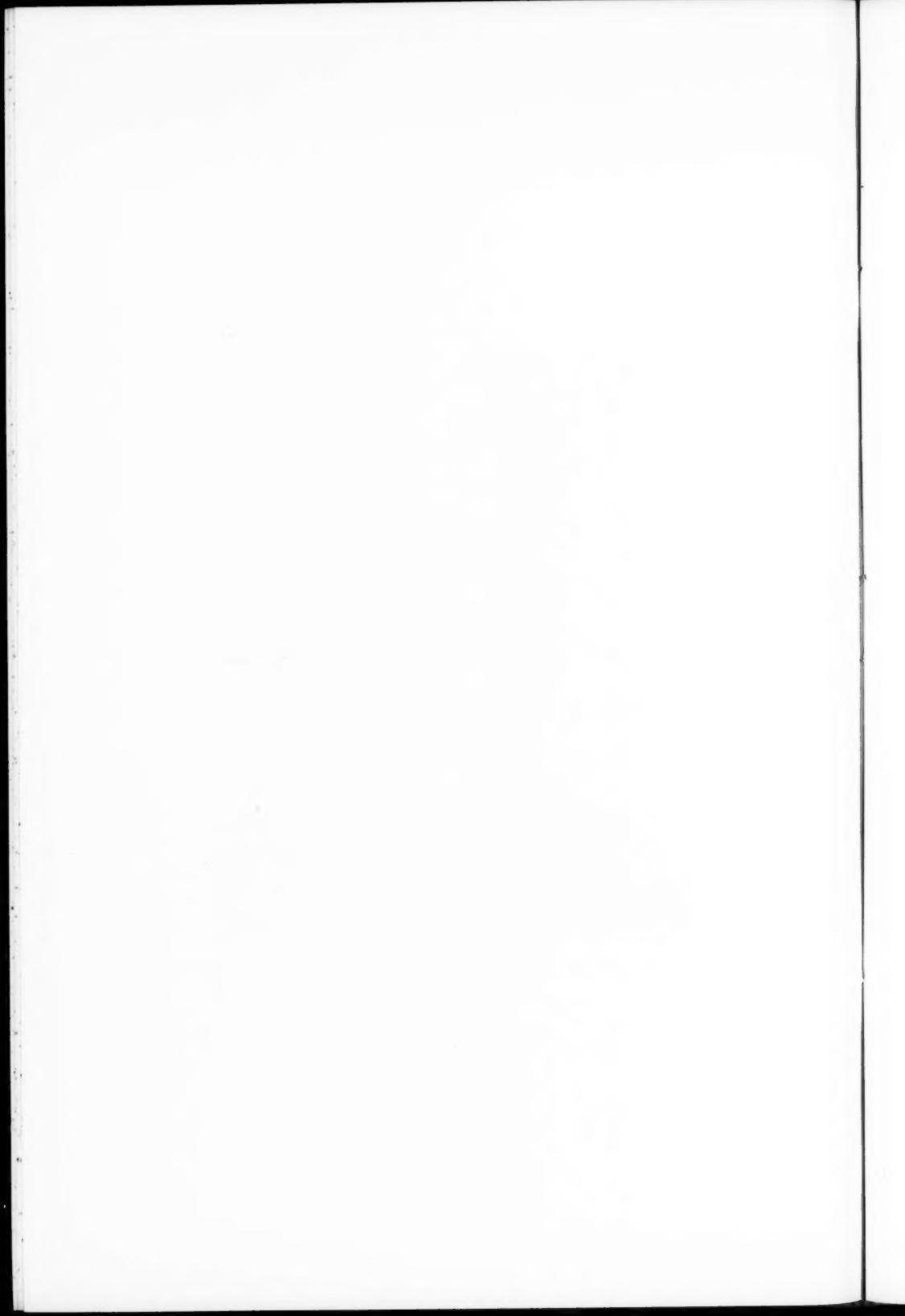
LXXII.

A CASE OF EPITHELIOMA OF THE ESOPHAGUS WITH SOME UNUSUAL FEATURES— SPECIMEN.*

By Samuel McCullagh, M. D.,

New York.

T. P., aged 57, consulted me on March 28, 1923, with the following history: About a year ago he first noticed a feeling as though there was an obstruction in his throat which interfered with swallowing. There was no pain, tenderness or interference with breathing. About nine months later he commenced to cough and raise thick tenacious mucus. About a month later blood appeared in the mucus in small amounts. This hemoptysis persisted for about a month, but was entirely eliminated by treatment. No history of hematemesis or regurgitation of food.

His principal complaint was the cough, which was exhausting on account of the tenacity of the discharge and the consequent difficulty in raising it. There was no pain on swallowing, though the difficulty persisted. On quiet breathing a distinct rattling was audible and marked dyspnea occurred on slight exertion. Has had a Wassermann test, which was negative. The sputum has been examined several times but no tubercle bacilli have been found. Has lost about 50 pounds in weight. An X-ray of the chest taken about a month ago was negative. Examination of the larynx showed a complete paralysis of the left cord, which was fixed, and a partial paralysis of the right cord which, on abduction, reached the cadaveric position. The whole larynx was congested but there was no evidence of new growth or ulceration. There was no marked adenopathy, though some enlarged glands could be felt below the larynx.

Dr. J. B. Solley, Jr., who examined his chest, reported: "The examination of the chest, on account of the stenosis, is

*Presented at the Forty-fifth Annual Congress of the A. L. A.

difficult. Percussion note is fair all over; there is no marked dullness or flatness. No rales. He coughed up a large 'gob' of thick yellow mucopus; there is therefore a raw surface somewhere in bronchi or alveolar spaces."

Dr. J. M. McKinney went over him from a neurologic standpoint and reported: "The neurologic examination, with the exception of the throat condition and a bilateral diminution of hearing, is negative. There is, apparently, a mass in the upper right mediastinum and some enlarged glands at the base of the neck. Pressure from this on the trachea might account for his breathing, but whether not it will explain the laryngeal paralysis I do not know."

Fluoroscopic examination with barium mixture showed some slight interference just above the clavicle, which apparently was confirmed by a plate, though this was very poor, and the interpretation was only made definite after the postmortem examination.

On April 6th, the patient's wife telephoned that he was choking to death, and he was ordered sent to the Manhattan Eye, Ear and Throat Hospital, where an emergency tracheotomy was performed by Dr. Robert Buckley. On opening the trachea two masses of tumor were coughed out, the larger piece being about 5 mm. by 3 mm., and the smaller about 3 mm. by 2 mm. A ragged tumor which bled very easily was seen on the posterior wall. The lumen of the trachea was so obstructed by this mass that Dr. Buckley inserted a piece of rubber tubing, as the tracheotomy tube that could be used was not long enough to go below the mass.

I saw the pateint about an hour later, and though his condition had greatly improved, respiration was still much embarrassed. He was again taken to the operating room, where I removed the rubber tubing and inserted a metal tube, after establishing the lower limit of the tumor by means of a bronchoscope introduced through the tracheotomy wound. The patient sank rapidly and died the next day.

Permission for a postmortem was limited to enlarging the incision. Following is the report submitted by Dr. A. A. Eggston, pathologist at the Manhattan Eye, Ear and Throat Hospital:

"The body is that of a well developed but poorly nourished white man. In the anterior midline of the neck, below the thyroid cartilage is an incised tracheotomy wound.

"In dissecting the tissues of the lower neck and upper mediastinum a dense infiltrating tumor mass is encountered. This mass lies between the esophagus and trachea and extends laterally, filling the entire upper mediastinum. The bronchial and mediastinal lymph nodes are enlarged and very firm and involved by the tumor mass. The esophagus, trachea and tumor mass is removed in toto.

"Upon opening the esophagus the entire upper one-third is indurated and shows an ulcerated area 10 by 6 mm. and covered with friable masses. In the lower part of the esophagus there are several tumor metastases upon the mucosa.

"Upon opening the trachea there is found a sessile oval tumor 4 by 2 cm., attached to the posterior wall. This mass is friable and hemorrhagic. The mass is 3 cm. above the bifurcation of the trachea. The tumor mass, between the esophagus and trachea, is very firm and measures 11 by 5 cm. The mediastinal nodes are likewise indurated.

"Microscopic examination of the esophagus and tracheal tumors and of the tumor proper show irregular areas and masses of rather large epithelial cells. The cells form no normal histologic structures and many show active mitosis. Many areas of necrosis are present in the epithelial cells. These sections are similar to the ones made from the tissue which was expelled by coughing before death.

"Diagnosis: Medullary carcinoma."

The points of particular interest to me are: (1) The friability of the tumor, the detachment of at least one of the fragments expelled at the time of the tracheotomy undoubtedly having been responsible for the sudden attack of asphyxia, and (2) the slight subjective symptoms until just prior to the end.

LXXIII.

SURGICAL DIATHERMY BY THE NEEDLE METHOD.*

BY ALFRED LEWY, M. D.,

CHICAGO.

Diathermy, thermopenetration or electrocoagulation, as it has been variously named, depends upon the heating of the tissues by their resistance to the electric current. For this purpose the high frequency (modification of the D'Arsonval[1] current is used, the interruptions, or rather alterations, being so frequent that they do not arouse nerve impulse or muscular contractions except when through poor contact sparking takes place, or the frequency is lowered otherwise. For surgical purposes a large indifferent electrode and a small one, or a needle inserted into the tissues, is used, so that where the current is concentrated around the small electrode intense heat is generated, causing rapid coagulation of the tissues—in fact cooking them. A number of works, [1] [2] [3] [4] describing various appliances, operating technic for different conditions, etc., have been published, so it is my intention to describe only a few additional observations of my own.

This subject seems first to have attracted attention to any extent as a therapeutic resource about 1909, when articles by Von Berndt[5] and Laqueur[6] appeared, followed shortly by Nagelschmidt,[7] who has since done a great deal of work on the development of apparatus and its clinical application, and probably introduced it to the British[8] in 1910, also writing a textbook (Lehrbuch) on the subject in 1913. Its surgical application, particularly in malignant growths, was thoroughly worked out by Doyen,[9] who probably deserves the greatest credit for work in this connection, although Cumberbatch[10] reports cases treated as far back as 1910.[11] Many others appeared in increasing numbers in foreign literature. In our country Clark, who refers to it as "dessication,"[12]

*Candidate's thesis. American Laryngological, Rhinological and Otological Society.

Corbus,[13] DeKraft,[14] Kolischer and Eisenstaedt[15] and Plank,[16] have done considerable work with this procedure in various parts of the body, and Novak[17] especially in the larynx.

It was from Dr. T. Howard Plank that I learned the technic which I use, namely, the needle in nearly all cases, because, as I believe, of its wider and more accurate application, the reduction in the number of electrodes, and the possibility of developing a more uniform technic.

The first essential is a machine which will deliver a current of just sufficient voltage to insure its traversing the entire body, or as much thereof as lies between the two electrodes, and not so high that there is great difficulty in insulating the cords and electrodes with which we have to work. The amperage must be sufficient to generate enough heat at the small electrode to coagulate the tissues immediately surrounding it. In working in the mouth and throat 500 to 1,000 milliamperes are usually sufficient, although I have used higher, and higher amperages are required for larger masses of tissue. The modern machines, of which there are a number on the market, are capable of fairly smooth control from small amounts of current for diminutive lesions, for instance, about the eye, up to five or more amperes. In this we have quite an advantage over the men who had to work with less flexible and convenient apparatuses, and who still achieved some excellent results. A good foot switch, under immediate control of the operator, and well insulated cords and electrodes to prevent unexpected shocks to the operator and undesirable burns to normal tissues, are indispensable. Most men use a large electrode, at least eight by six inches, covered by towels or some special tissue, wet with 5 to 10 per cent salt solution. This must be kept uniformly wet, as if it dries out in places, all current passes through the remaining wet area, and may be sufficiently concentrated to cause a burn. As the electrode dries out rapidly under the heat generated by the current, this is an important thing to watch. By using an uncovered block tin electrode, carefully applied to the body, having it bandaged on and the patient lying on it, the connecting wire hooked into an eyelet, this part carefully protected, much of the danger of a burn can be obviated. The operating needle, which should be in a well insulated handle, may be of any

desired length with sufficient strength, as it can be covered
with a rubber catheter, exposing only so much as the depth
we wish to penetrate in order to cause the right amount of
coagulation. Various metals have been used, but Dr. Emil H.
Grubbe has suggested to me iridioplatinum (commonly used
with the galvanic current) as best fulfilling the indications.
Other operators seem to prefer disc, button or ball electrodes,
and in some cases a knife for excising and coagulating at the
same time. These are all described in the textbooks men-
tioned above. The needle is inserted before switching on the
current, which is again turned off before removing the needle,
otherwise sparking takes place, causing muscular contraction
or a burn where it is not wanted. This sparking, however, can
be turned to good use in stopping bleeding by charring the
surface.

The special advantages of the needle are: That it does
away with a considerable number of special tips; that they
can be placed accurately, but particularly that it can be
made to traverse several layers of tissue, through fascial planes,
with a greater probability of accuracy in the amount and depth
of tissue coagulated. This is well shown in figure 1, photo-
graph of a piece of meat, in which four punctures were made.
Each one was given twenty seconds' application of 700 milli-
amperes. The similarity of all except one will be noted, which
shows coagulation extending at an angle from the tip. This,
I believe, was caused by tilting the needle in situ, separating
the muscle bundles and permitting the steam generated in the
moist tissues to travel along the separation. I shall refer to
this steam production further on.

Figures 2 and 3 show a piece of meat composed of two
muscle layers, separated by fascia, which were treated by a
disc electrode (the method mostly used by others). Figure 2
shows the surface, figure 3 a cut through one of the coagu-
lated areas. Figure 4 the same, enlarged three diameters. The
applications were also twenty seconds each, of 800 milliam-
peres. In these pieces the surface coagulation is wider, but
the depth is not so great. The picture does not show it clearly
but it does not extend quite down to the fascia, although the
total thickness of the meat was slightly over one inch, as
against two inches. If we can draw any conclusions from

these two experiments, it would seem that the disc electrode would be useful for more superficial work in homogeneous tissue, while the needle should be preferred for deeper application and where more than one layer of tissue is to be coagulated. The needle can take the place of the surface electrode by making a shallow insertion.

Most writers advise that the application be stopped as soon as steaming is observed, so as to avoid sparking, which occurs as soon as an area becomes carbonized, and thereby a poor conductor, whereupon the heating lessens, and theoretically the coagulation in the deeper parts ceases. While this is relatively so, I do not think it is so important when using the needle, as I have observed the coagulated area to extend even after the needle track is carbonized and sparking begins. The question of steam production in the tissues is, I believe, an important one, and one that does not seem to have received sufficient attention. It is difficult to calculate along what planes the steam will travel, and the steam, of course, cooks the tissues as well as the heat from resistance to the current. A detailed knowledge of the anatomy of the parts may be of help, and undoubtedly we will learn from experience. While the circulation of the blood in living tissue tends to dissipate the heat, the presence of greater moisture permits the coagulation process to become wider before carbonization, and this fact is taken advantage of by some,[18] who drop salt solution on the part on which they are working. It also leads to greater steam production, which, as pointed out above, is one of the factors which prevents accuracy in localizing the destruction of tissue, as desired.

The proximity of the large vessels in the neck, and of important nerves, particularly the pneumogastric, must always be kept in mind in working in this region. To some extent the blood vessels are protected by the rapid flow of blood through them, acting much as does the water in an automobile radiation system in dissipating the heat. In cases 1 and 2, described by me, I inserted the needle behind the tonsil as is done in block anesthesia for tonsillectomy. I realized that I was taking chances, but believed it was justified by the almost certain fatal termination, if the growth was not entirely destroyed. Fortunately there was no secondary hemorrhage.

although this has occurred in some cases. Preliminary liga-
ture, especially of the lingual, has been advised. I am not
sure as to the advisability of this in the case of the carotid
and jugular, as their circulation probably also protects to some
extent the pneumogastric nerve. If a malignant growth has
penetrated to this depth it is probably hopeless except for tem-
porary relief at any rate. Because of the outpouring of fluid
into the tissue adjacent to the coagulated area, operations in or
about the larynx require tracheotomy. Where dessication
(fulguration) alone is used, this will probably not be neces-
sary. I am using the term dessication in preference to ful-
guration, because dessication describes what takes place in
the tissue when the spark is used. This process is useful for
superficial or diminutive lesions, and has the advantage in skin
lesions of leaving a smooth white scar, where the coagulation
caused by actual contact of the electrode may cause keloid.

The advantages and disadvantages of this method of sur-
gery are well summed up by Saberton :[19]

1. Tumors otherwise inoperable may be attacked.

2. The operation is more or less bloodless.

3. The danger of metastasis is much less than with a cut-
ting operation, owing to the sealing of the blood vessels and
lymphatics draining the parts.

4. Sterilization of the parts.

5. There is complete destruction of the visible and palpable
malignant disease.

6. The operation is rapid and easy.

7. There is no surgical shock.

8. Convalescence is rapid.

9. Formation of adhesions is rare.

10. The operation can be repeated if necessary.

Disadvantages :

1. Healthy and diseased tissues are equally destroyed.

2. The surgeon cannot see important vessels and nerves.

3. There is danger of secondary hemorrhage when operating
near large blood vessels.

4. There is a tendency to formation of keloid in operations
involving skin surfaces.

To this last I may add that in operating near bone, destruc-
tion of the periosteum may lead to long continued suppuration.

To No. 7, I may add that while surgical shock may usually be negligible, the necessity of using chloroform as an anesthetic increases the anesthetic risk. Ether can be used by first giving morphinscopolamin enough to have the patient quite stuporous, then using just enough ether to complete anesthesia, and before beginning the diathermia removing the ether from the room. I have heard of one case of ether explosion caused by this method, and in one of my cases prolonged cyanosis, supposedly due to the heavy dosage of morphin and hyoscin, caused a great deal of anxiety. The disadvantage No. 1, "Healthy and diseased tissues are equally destroyed," is not absolutely accurate, as Doyen has shown[20] that the malignant cells succumb to a somewhat lesser degree of heat than do mature cells.

I believe that diathermy presents distinct advantages over any other method for removal of malignant growths in the mouth and throat, and for that matter in several other regions, and have therefore taken the liberty of adding my few observations to the fundamental work already done by others. The removal of malignant growths by this method, as with other methods, should be supplemented by subsequent raying, by removal of gland bearing tissue where necessary, and by the use of such measures and remedies as experience has apparently proven useful. With potassium nitrate intravenously, claimed by some to relieve the pain of inoperable cancer, I have had but one experience, and that not favorable. Arsenic in tonic doses I have used a number of times, but cannot yet present any definite data concerning it.

Case 1.—Male, age 55. For over two months complains of severe pain in right side of throat. Swallowing difficult; has lost considerable weight and strength; looks cachectic. Examination shows ulcerative process involving right tonsil, both pillars in part and the adjacent part of the tongue; induration extends toward angle of jaw; no perceptible cervical adenitis. Section removed shows squamous celled epithelioma. Operation: Electrocoagulation (diathermia) by the needle method, destroying all the involved tissue. Patient was free from pain immediately following operation, and ate in comfort for the first time in months. Most of slough separated in two weeks, but healing was delayed by a splinter of bone from ramus of

mandible which had evidently been overheated and which was extruded in three weeks. This patient had no dissection of glands of neck and no raying (this was four years ago). He remained well, but nine months later a swelling appeared in infraclavicular region which eventually resulted in death. There was no recurrence in situ.

Case 2.—Male, age 53. For three months has had pain in right side of throat and tongue. Examination reveals ulcerative process on the right side of the tongue, right tonsil and both pillars, with quite extensive induration toward base of tongue and into floor of mouth. Section made at time of operation showed squamous celled epithelioma. Attempt was made to coagulate the entire area, and block dissection of neck followed, but healing did not take place, and there was further invasion of contiguous tissues. Another but unsuccessful effort was made to destroy the invaded area. This patient had no relief, even temporary, from the operations and eventually died.

Case 3.—Male, age 66. Has had pain in throat and up into head for three months. Examination shows an ulcerative process involving left tonsil, both pillars, soft palate and posterior pillar of right side. There was very little induration, and smears showed fusiform bacilli and spirilli in profusion. He was treated for a brief period for Vincent's angina, then section was made, which showed the process to be a squamous celled epithelioma. Two months were lost in radium applications, under which the process progressed. With diathermy the entire involved area was coagulated, including soft palate, and, after the slough separated, the original area looked well, but there was no relief of pain, which was severe, and shortly there appeared new nodular areas in the nasopharynx, which, because of the absence of the soft palate, were easily reached for another attempt at coagulation. There was no relief and soon after the second operation the patient went into stupor and died two weeks later. In this case I believe the brain was invaded, as no urinary or other findings explained the stupor.

Case 4.—Male, for two months has had pain on swallowing, located on right side of tongue. Has lost weight and strength, probably largely from lack of nourishment, as, although he

looked badly, there was no definite cachexia.   Examination showed deep fissure in right side and dorsum of tongue, opposite the tonsil, with two fungating nodules also on dorsum. The posterior pillar appeared to be slightly infiltrated.   Section taken at time of operation showed the process to be a squamous celled epithelioma. The posterior pillar, part of tonsil and an area of tongue extending beyond all the involved area were coagulated.   There was no relief of pain until the slough separated, about two weeks later. This operation was followed by block dissection of the right side of neck and a course of X-ray. Following the first X-ray treatment there was a large infiltration of the tissues of the neck, which entirely disappeared in three months. This patient was also put on a colloid preparation of arsenic in tonic doses for nearly a year. It is now (October, 1922) over two years since the operation and the patient is well, has gained in weight and strength and, since three months after the operation, has been working as usual. The tongue was quite stiff for a while, but is now more movable. It can be protruded just beyond the teeth. There has been an annoying paresthesia of the right side of the tongue, which still persists, and about the right one-third is also anesthetic. The defect in the side of the tongue left by the operation is plainly seen.

NOTE.—Patient No. 4 is still living and well.

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110 N. Wabash Ave.
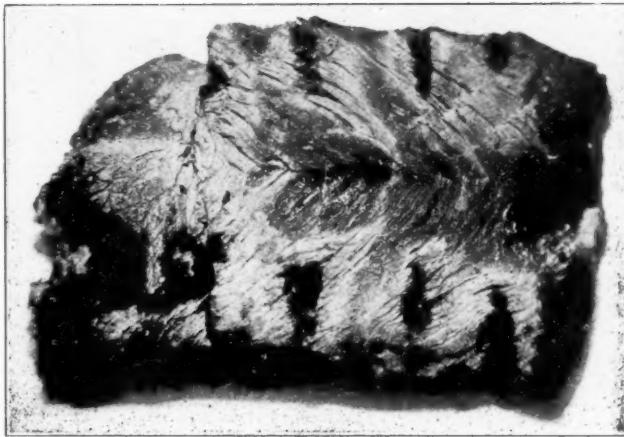
FIGURE 1.

Piece of meat, two inches thick; four punctures, 20 second applications of needle at 700 milliamperes. Cut through punctures and laid open. Notice how coagulation tends to follow the "grain" of the fibre bundles; probably due to steam. Otherwise the extent of coagulation is quite uniform.
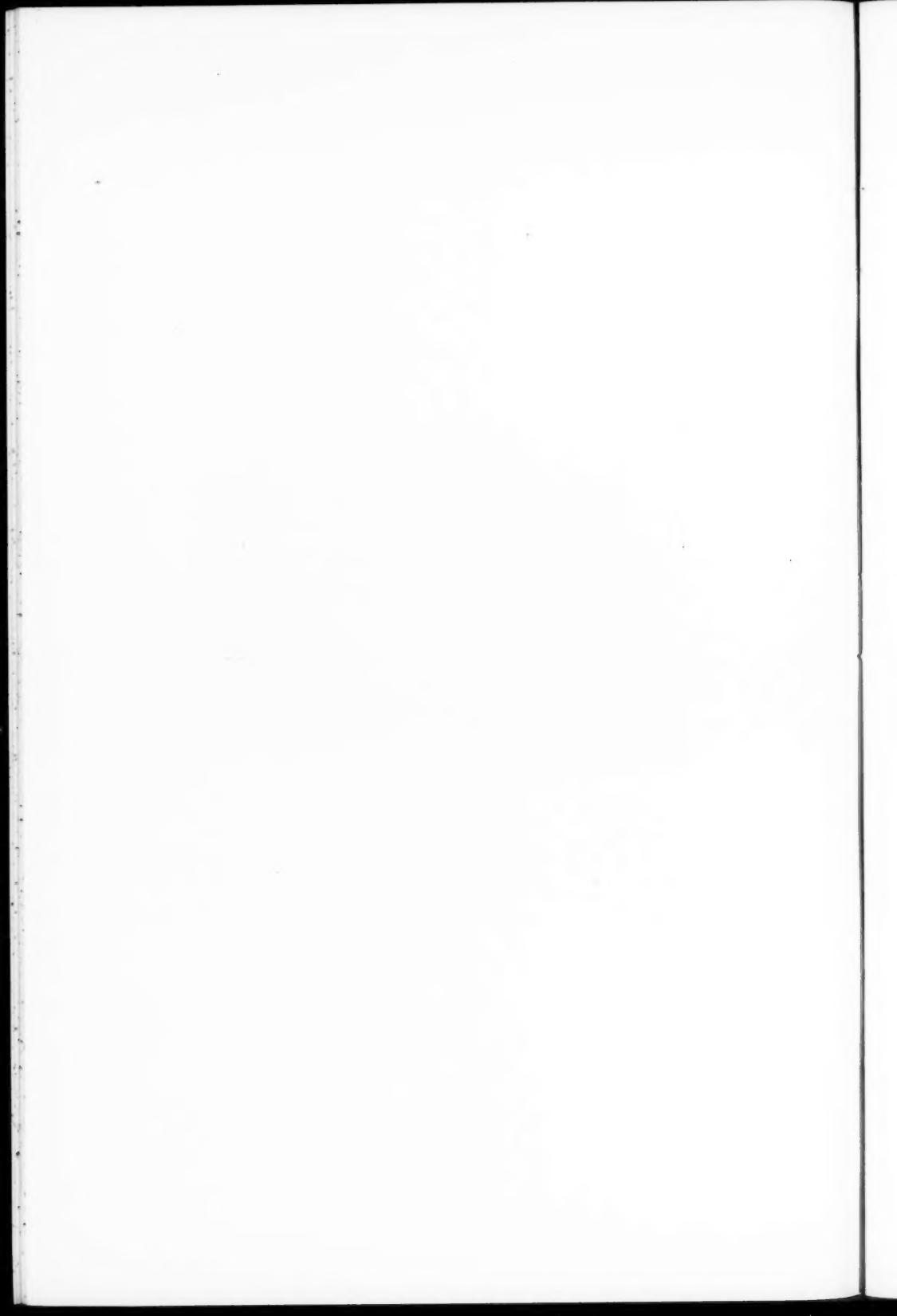
FIGURE 2.

Showing size of surface coagulation with flat electrode 6 mm.
diameter, 20 seconds' application, 800 milliamperes.
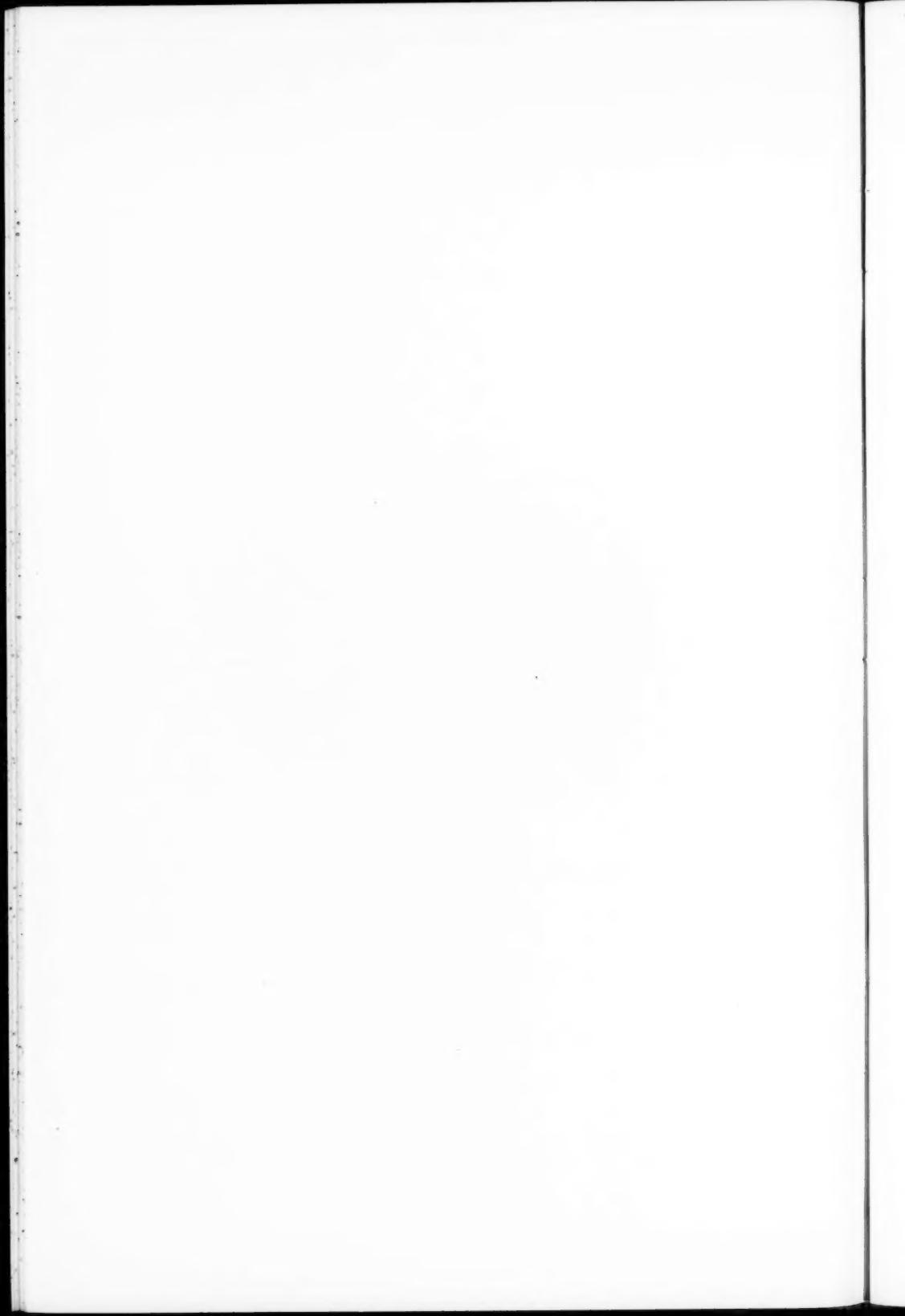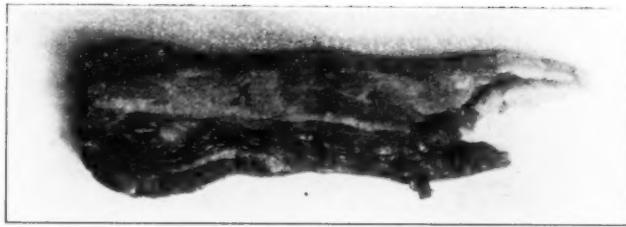
FIGURE 3.

Same piece as Fig. 2, on cut section. The coagulated area, marked X, appears to reach the fascia, which in reality it did not quite do. The surface at the point of section is at the top of the coagulated area. The meat behind it is pushed up a little higher, and appears higher on the picture.
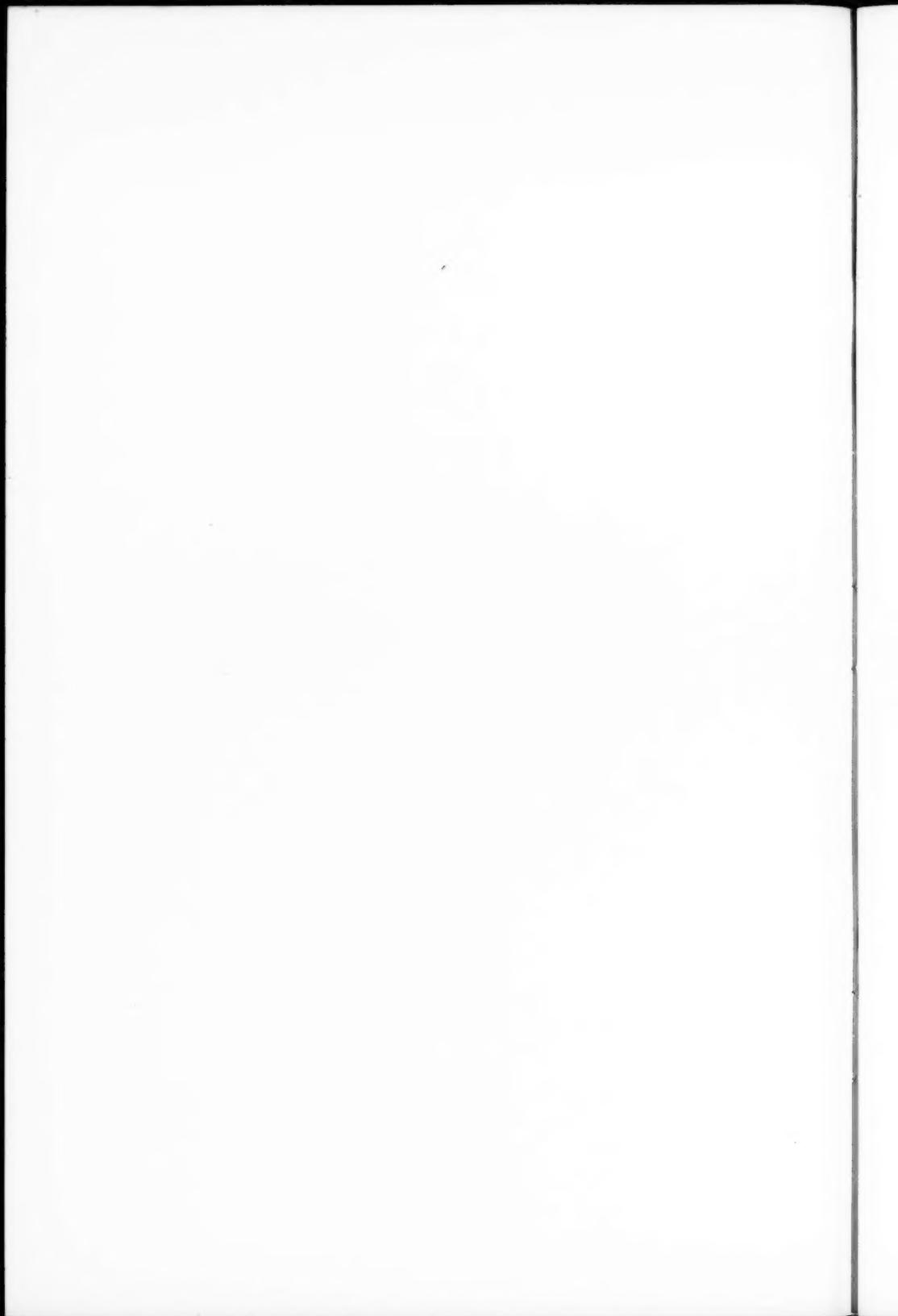
FIGURE 4.

The same as Fig. 3, magnified about two diameters. To the left of the coagulated area is fat, and below it is fascia showing white. The coagulated area appears gray.
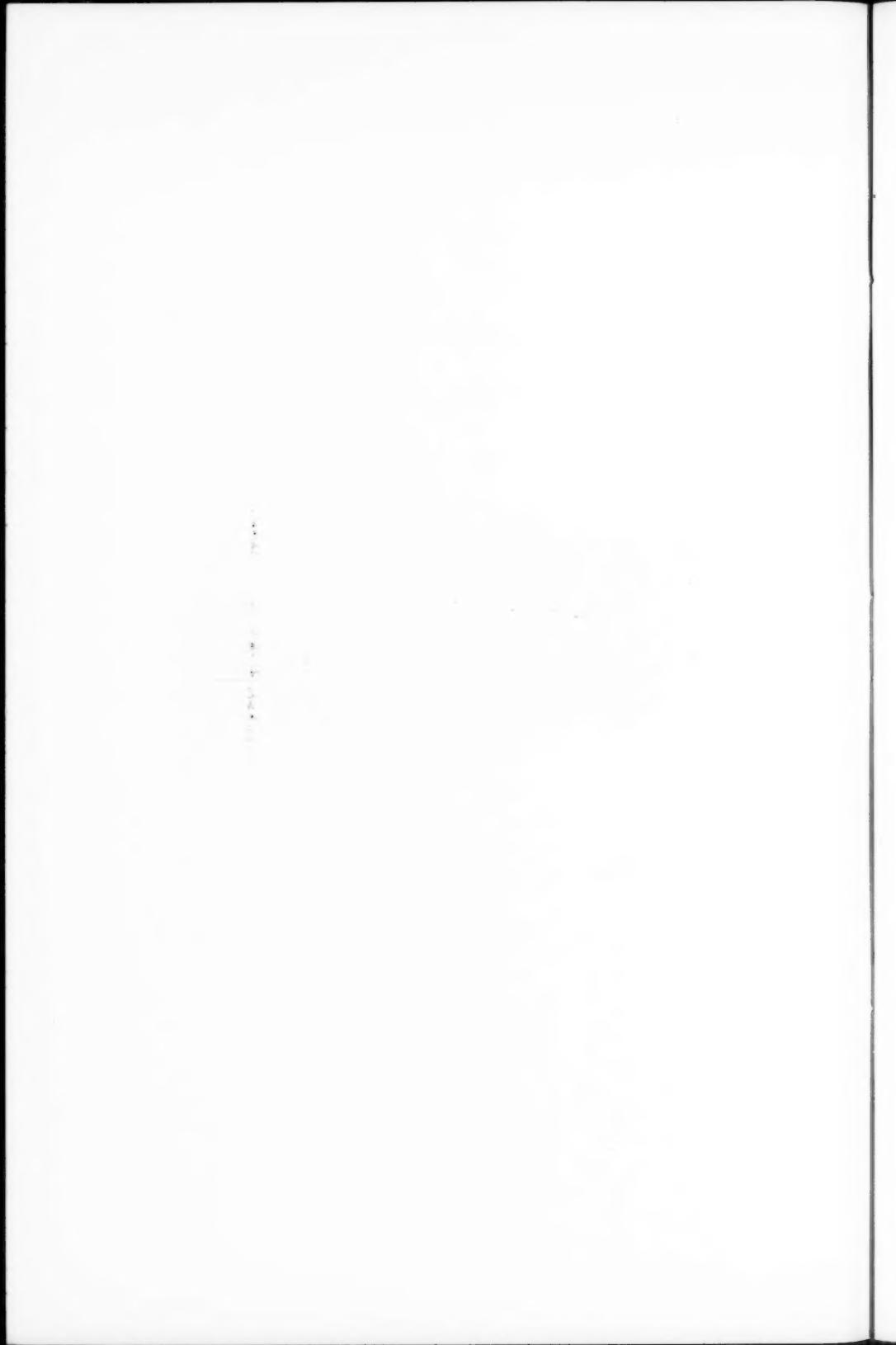
LXXIV.

FURTHER STUDIES OF FATALITIES FOLLOWING OPERATIONS ON THE NOSE AND THROAT.*

By H. W. Loeb, M. D.,

St. Louis.

In a paper read before this Society a year ago, I presented a study of 332 hitherto unreported fatalities following operations upon the nose and throat.

Since that time I have collected 112 more, making a total of 444 cases which constitute the basis of this paper. How many more there are which have thus far eluded this inquiry is a matter of conjecture, though it is likely that the number is not inconsiderable.

It has been my experience that, whenever this subject is brought up at medical conventions or privately among several laryngologists, quite a number of unincluded cases have been added.

A summary of the grouping of the cases, according to the previous and present reports, yields the following:

	Previous	Additional	Total
1. Meningitis	124	44	168
2. Hemorrhage	55	21	76
3. General sepsis	20	7	27
4. Erysipelas	8	1	9
5. Endocranium (excluding meningitis)	27	4	31
6. Respiratory tract	43	12	55
7. Heart	5	0	5
8. Miscellaneous	20	16	36
9. Undetermined	30	7	37
Total	332	112	444

It is to be noted that there has been no material change in the predominance of one type of fatality over another. However, there has been an increase of 35 per cent in the reports of meningitis and 40 per cent in those of hemorrhage, although the total number of cases added is only 30 per cent. As in the previous report, care has been observed to exclude

*Read before the meeting of the American Laryngological, Rhinological and Otological Society, May, 1923.

cases, the history of which was incomplete, doubtful or inexplicit.

I. MENINGITIS.	Previous	Additional	Total
1. Tonsillectomy	4	3	7
2. Intranasal frontal operation	16	3	19
3. Probing and irrigating frontal sinus	7	0	7
4. Ethmoid operation, with or without resection of the middle turbinate	39	23	62
5. Sphenoid operation	10	0	10
6. Maxillary operation	1	1	2
7. Resection of middle turbinates	15	5	20
8. Submucous resection	13	2	15
9. Removal of polypi	13	2	15
10. Combined sinus operation	1	3	4
11. Combined submucous and sinus operation	5	2	7
Total	124	44	168

Three cases of meningitis following tonsillectomy have been added to the list. Of the 41 others, 23 are ascribed to ethmoid operations, making a total of 62, and 18 fatalities were due to operations including or adjacent to the ethmoid cells.

II. HEMORRHAGE.	Previous	Additional	Total
1. Tonsillectomy (with or without coincident adenoidectomy	43	19	62
2. Adenoidectomy alone	2	0	2
3. Incision of peritonsillar abscess	3	0	3
4. Submucous resection	2	0	2
5. Sinus operation	4	2	6
6. Resection inferior turbinate	1	0	1
Total	55	21	76

The increase has been almost exclusively due to tonsil operations.

III. GENERAL SEPSIS.	Previous	Additional	Total
1. Intranasal maxillary operation	2	0	2
2. Submucous resection	1	2	3
3. Tonsillectomy	16	3	19
4. Incision peritonsillar abscess	1	1	2
5. Probing frontal sinus	0	1	1
Total	20	7	27

IV. ERYSIPELAS.	Previous	Additional	Total
1. Submucous resection	4	0	4
2. Sinus operations	2	0	2
3. Resection middle turbinate	2	0	2
4. Tonsillectomy	0	1	1
Total	8	1	9

The addition of a fatality due to tonsillectomy is to be noted.

V. ENDOCRANIUM, EXCLUDING MENINGITIS.

	Previous	Additional	Total
1. Brain abscess	5	1	6
2. Cavernous sinus thrombosis	9	3	12
3. Cerebral embolus or hemorrhage	13	0	13
Total	27	4	31

Three new cases of cavernous sinus thrombosis make the possibility of this complication a matter of some concern.

VI. RESPIRATORY TRACT.

	Previous	Additional	Total
1. Foreign body	5	0	5
2. Edema larynx and pharynx	6	0	6
3. Pulmonary edema	1	0	1
4. Pneumonia	17	5	22
5. Pulmonary embolus and abscess	14	7	21
Total	43	12	55

Reports of pulmonary abscess have been increased 50 per cent. We may be sure that there are many yet to come.

VII. HEART.

	Previous	Additional	Total
1. Angina pectoris	1	0	1
2. Dilatation	1	0	1
3. Asthenia	1	0	1
4. Infarct	1	0	1
5. Rupture	1	0	1
Total	5	0	5

VIII. MISCELLANEOUS.

	Previous	Additional	Total
1. Acidosis	1	0	1
2. Delirium tremens	1	0	1
3. Dilatation of the stomach	1	0	1
4. Diphtheria	1	1	2
5. Epilepsy	1	0	1
6. Hyperthyroidism	2	0	2
7. Orbit injury	1	0	1
8. Poisoning	1	0	1
9. Scarlet fever	3	0	3
10. Shock	1	3	4
11. Status lymphaticus	5	7	12
12. Uremic coma	1	3	4
13. Uterine hemorrhage	1	0	1
14. Convulsions	0	1	1
15. Acute lymphatic leukemia	0	1	1
Total	20	16	36

IX. UNDETERMINED.

	Previous	Additional	Total
1. Tonsillectomy	16	4	20
2. Adenectomy alone	1	0	1
3. Exenteration ethmoid, with or without removal of polypi...	4	1	5
4. Puncture of nasomaxillary wall, irrigation of the sinus...	5	1	6
5. Submucous	1	0	1
6. Resection middle turbinate.............................	2	0	2
7. Combined tonsillectomy and sub-mucous resection	1	0	1
8. Incision peritonsillar abscess....................	0	1	1
Total	30	7	37

Many of the cases reported carry with them important lessons of indication and contraindication, of inexpertness, of danger of multiple operations, of postoperative inattention and of unforeseen accident.

The following may be cited:

1. Incision of a supposed peritonsillar abscess followed by death from acute lymphatic leukemia.

2. Another from myelogenous leukemia after a tonsillectomy.

3. Acute nephritis following combined submucous resection and tonsillectomy in a patient suffering from nephrolithiasis.

4. Septicemia and phlegmon of the neck following combined submucous resection and tonsillectomy.

5. Tonsillectomy in a patient with hydrocephalus followed by meningitis.

6. Meningitis following resection of the middle turbinate; patient who left town on the same day, having a hemorrhage that night, had his nose plugged by inexpert physician.

7. Extensive removal of polypi followed by meningitis, autopsy showing that a portion of the cribriform had been torn away.

8. Cavernous sinus thrombosis following complete exenteration of both ethmoid labyrinths and resection of the anterior wall of both sphenoids.

9. Tonsillectomy in a patient who was intoxicated one day before the operation and four days after. Death from meningitis.

10. Three days after an acute cold patient developed severe pain in the face. Diagnosis pansinusitis. All the sinuses were opened intranasally on the same day. Patient conscious with a temperature of 102 degrees. After operation, patient lost consciousness and became comatose with a temperature of 106,° profuse discharge from the nose and symptoms of meningitis. Radical operation on sinuses on both sides performed but patient promptly died of meningitis.

11. Acute frontal sinusitis, anterior end of middle turbinate removed for drainage on account of pain. On the third day the patient died from meningitis.

12. Polypi removed with biting forceps, both middle turbinates resected and ethmoid cells, both sides, exenterated. Operator was able to see pulsation in region of cribriform plate with escape of cerebrospinal fluid. Death from meningitis in three days.

13. Fatality from meningitis on the third day after exenteration of both ethmoid labyrinths with Ballenger's knife.

14. Tonsillectomy in a patient who had had two attacks of Vincent's angina. Several days later cervical lymphatics became swollen and tender, with a temperature of 107 degrees. Incision resulted in the evacuation of a small amount of very offensive pus which contained fusiform bacilli and Vincent's spirilla. Patient recovered rapidly but ten days later developed pulmonary abscess from which he died.

15. Fatality from acute uremia following a local tonsillectomy in a marked nephritic.

16. Fatal meningitis following pansinus operation three days after onset of attack.

17. Tonsillectomy followed by death in 12 hours from hemorrhage in a hemophiliac.

18. Injury to the meninges in removing polypi, brain substance coming through the nose while the patient was lying in bed. Confirmed by autopsy.

The study of these cases has served to strengthen the views expressed in my previous paper and impels me to urge all who meet with these unfortunate experiences to insist upon autopsies and complete autopsy protocols, to the end that we may have a better understanding of the processes involved and better means of combating them.

LXXV.

THE ANATOMY OF THE SPHENOID FISSURE.*

By Greenfield Sluder, M. D.,

St. Louis.

In 1914† I injected the nasal (sphenopalatine, Meckel's) ganglion and paralyzed the abducens nerve. The abducens palsy recovered in three months. All diplopia vanished. I accepted this as the logical sequence of such an accident according to the accepted anatomic understandings of the anatomy of the sphenoid fissure—i. e., that the alcohol passed upward and outward from the pterygomaxillary fossa and reached the sphenoid fissure, meeting there the lowermost nerve—the abducens. The tissues were very loose in this case. The alcohol met with no resistance.

Five years ago Dr. Joseph C. Beck injected the nasal ganglion and paralyzed the oculomotor nerve, in a patient who later consulted me. This led me to think that the anatomy of the sphenoid fissure was subject to variations.

The accompanying drawings of dissections of the sphenoid fissure show that the positions of the nerves are not constant. The abducens is probably oftenest the lowermost. Sometimes, however, the oculomotor, if not in its full trunk certainly in some of its branches, is lowermost, which accounts for the clinical phenomena just mentioned.

FIGURE 1.

Bony environments of sphenopalatine foramen and sphenoid fissure, demonstrating their relations and the distance between them. The foramen rotundum also appears.
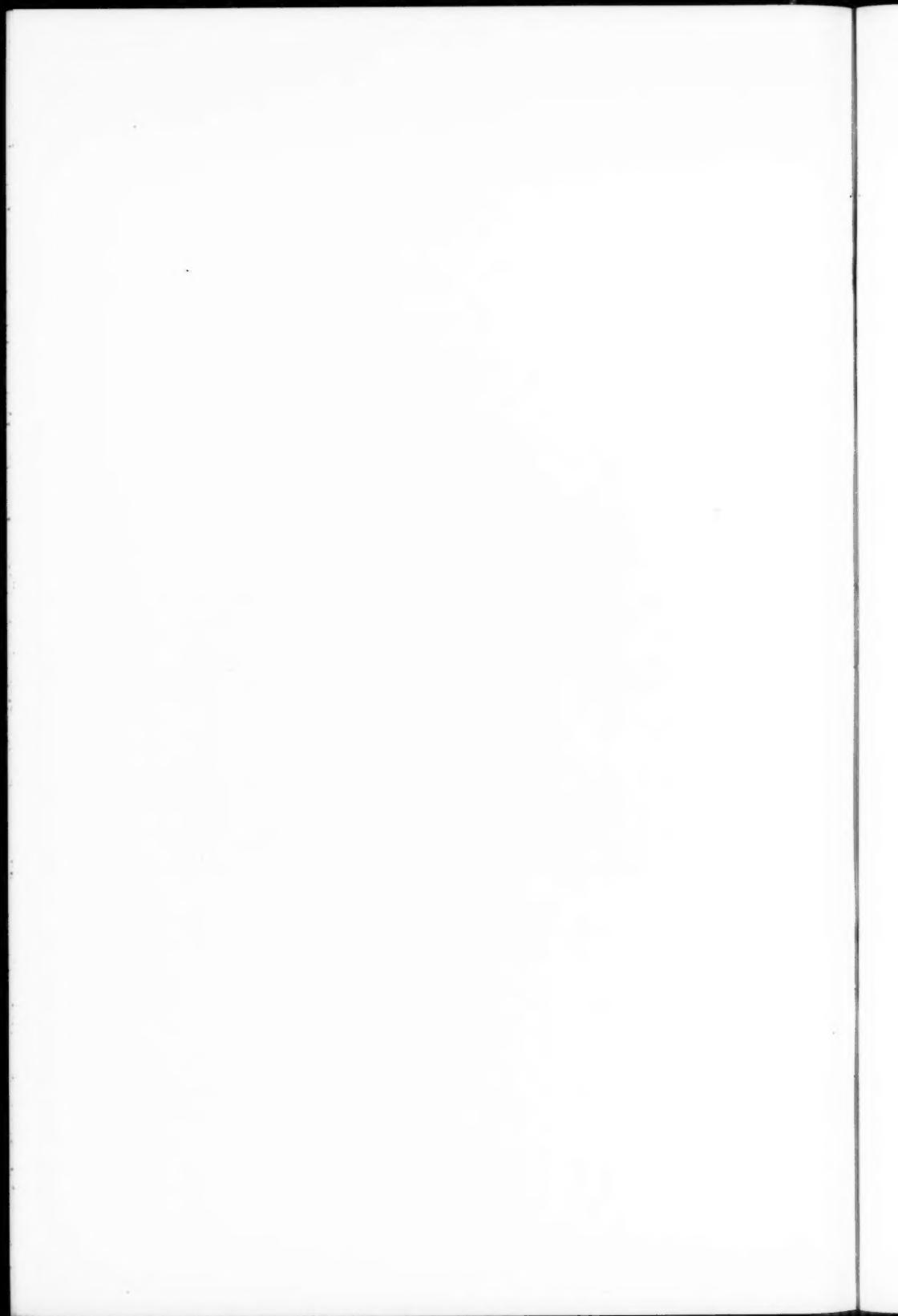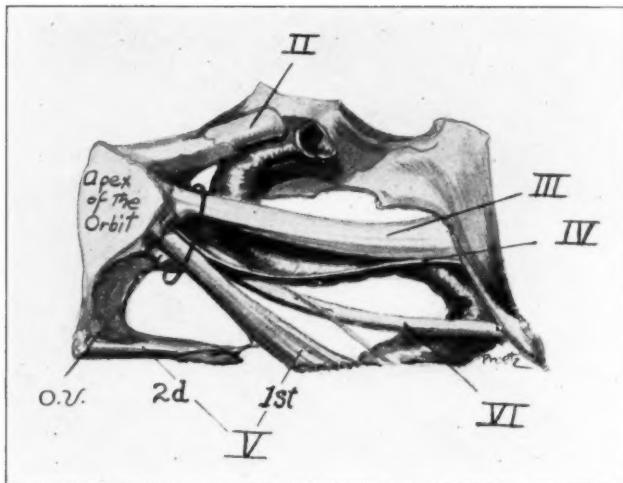
FIGURE 2.

Semidiagrammatic representation of the contents of the sphenoid fissure, showing their relations to one another, both in the fissure and in the cavernous sinus. Left side. The sixth nerve as it passes through the sphenoid fissure here lies mesial to the fourth and the first division of the fifth above the ophthalmic vein. Note the position of the internal carotid with relation to the other structures.
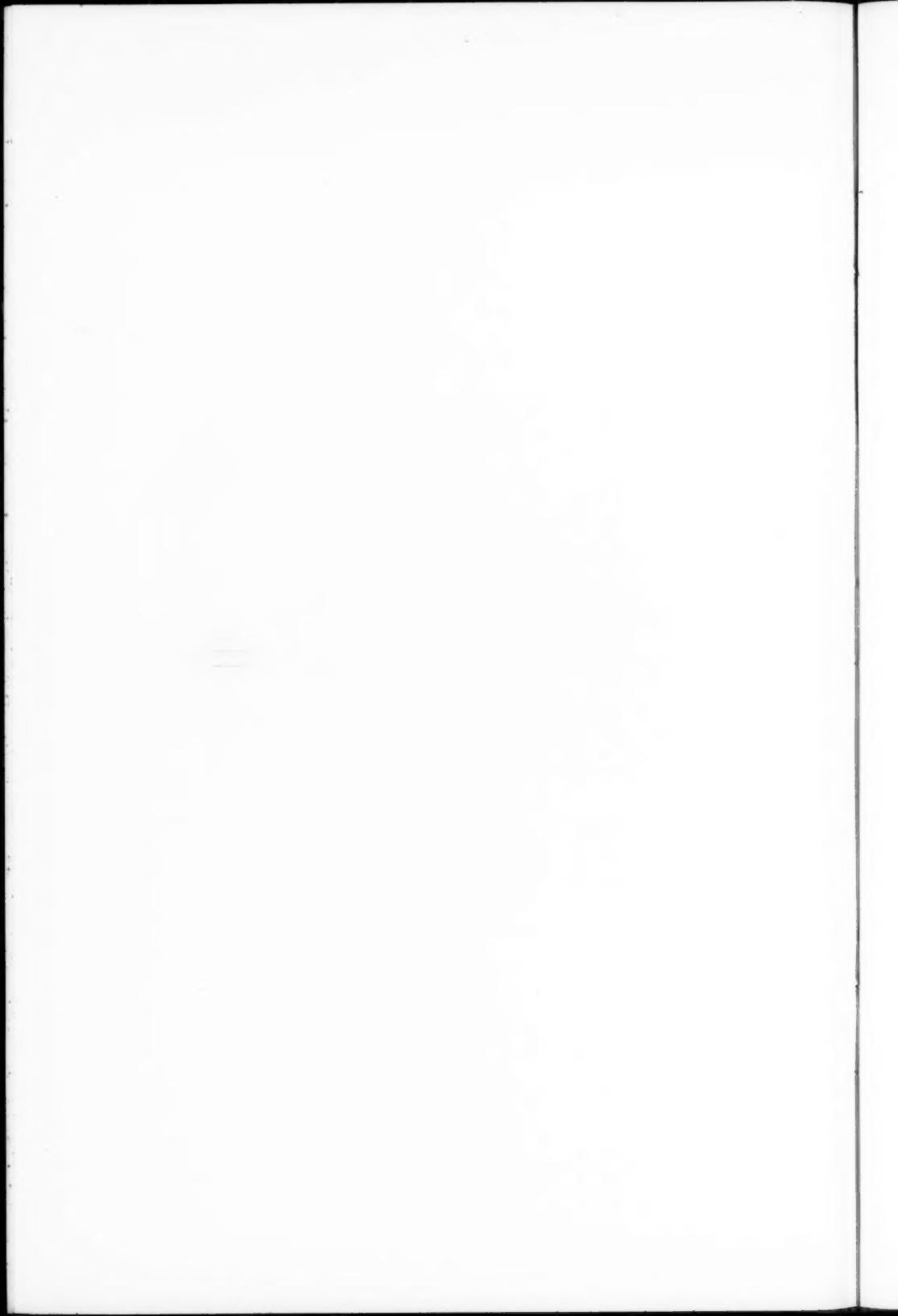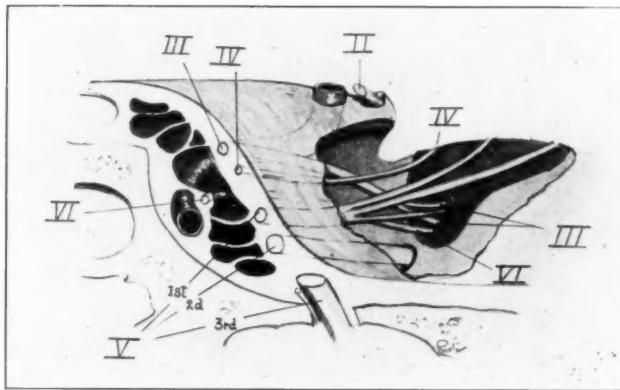
FIGURE 3.
Relations of the structures in the cavernous sinus and the sphenoid fissure as depicted in Cunningham's Anatomy.
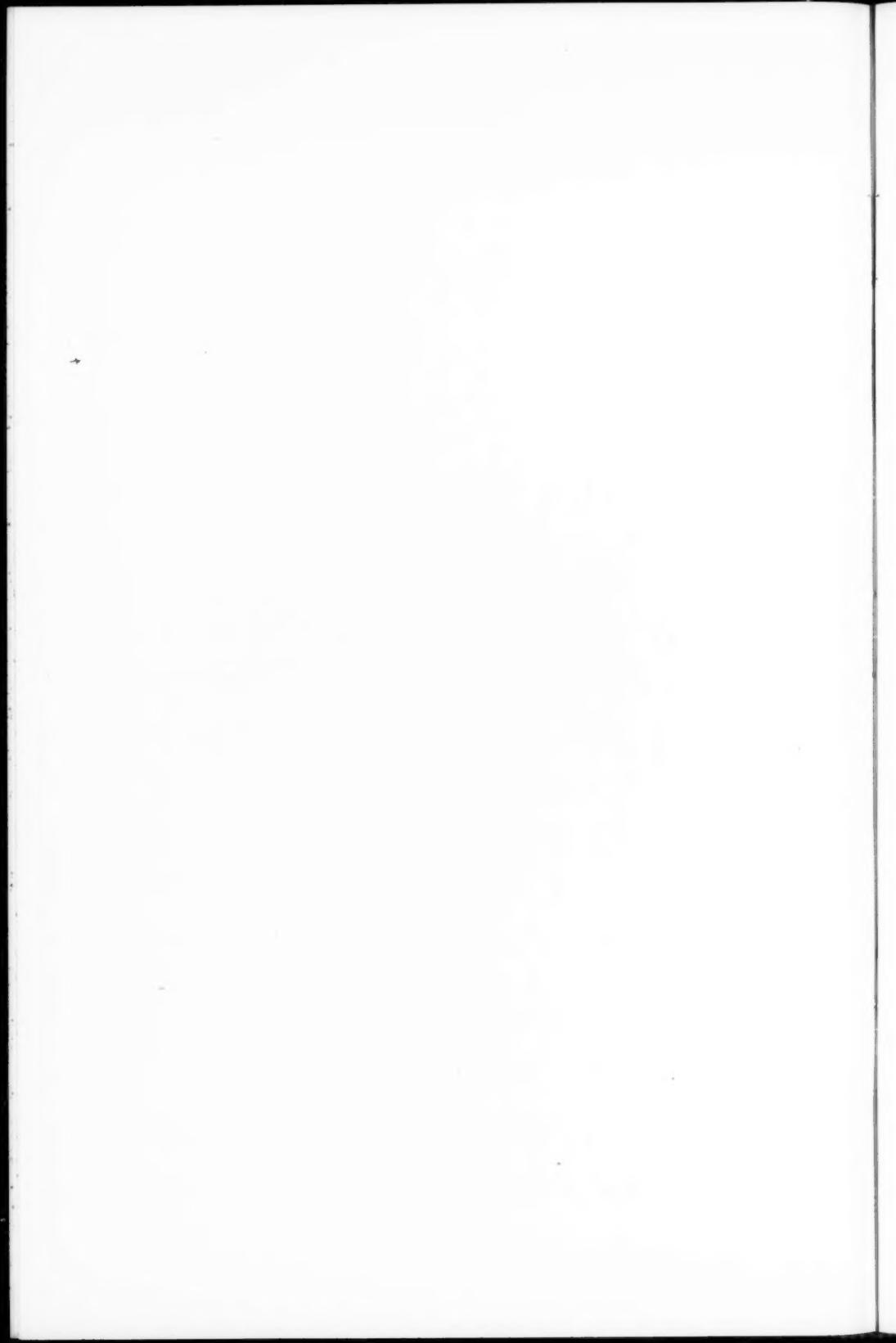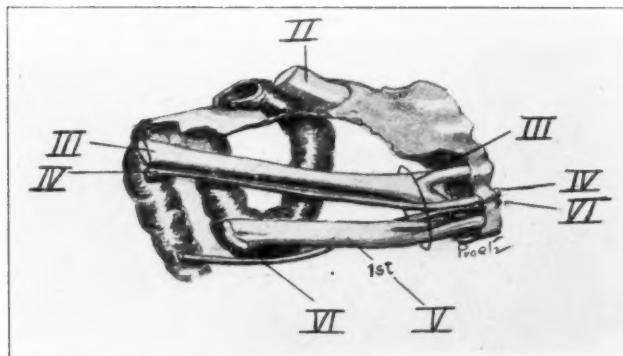
FIGURE 4.

Contents of the sphenoid fissure right side. In this case the sixth nerve passing through the cavernous sinus mesial to, and at a lower level than the first division of the fifth, ascends still mesial to it and traverses the sphenoid fissure above it and in conjunction with the fourth. Note the variation in plane and degree of tortu-csity of the internal carotid as compared with Fig. 2.
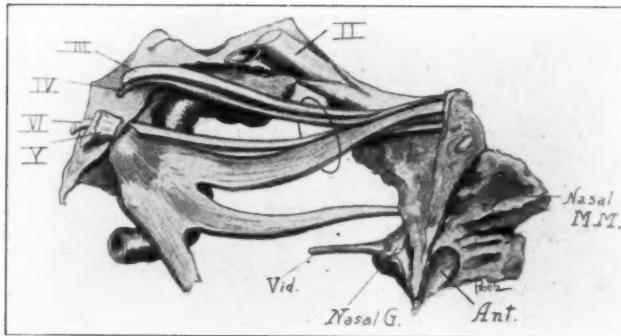
FIGURE 5.

The contents of the sphenoid fissure, showing their relations to one another and to the nasal ganglion and the vidian nerve. The intimate relation of the ganglion to the posterior superior extremity of the maxillary sinus is also shown.

Lateral view of structures on the right side.
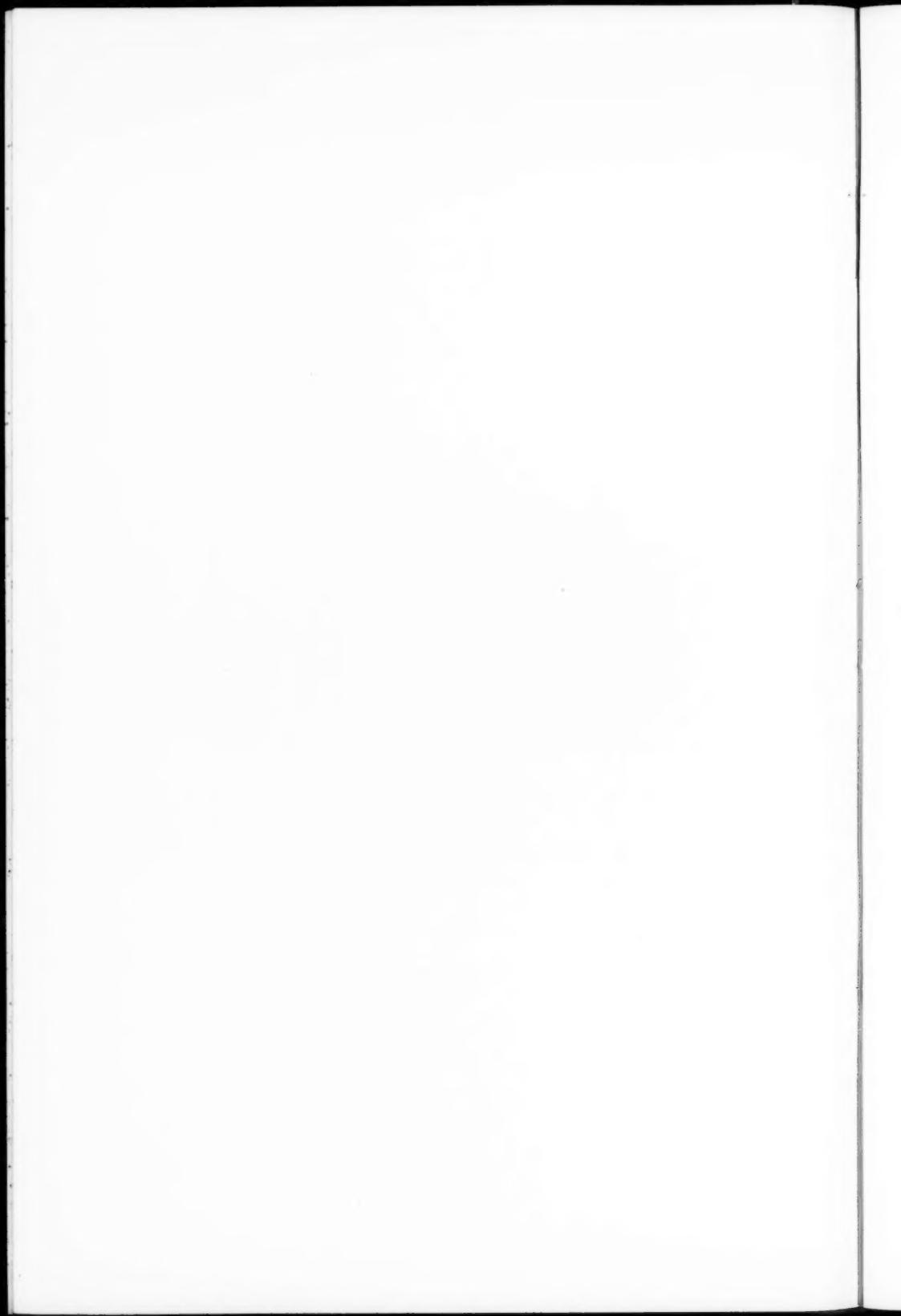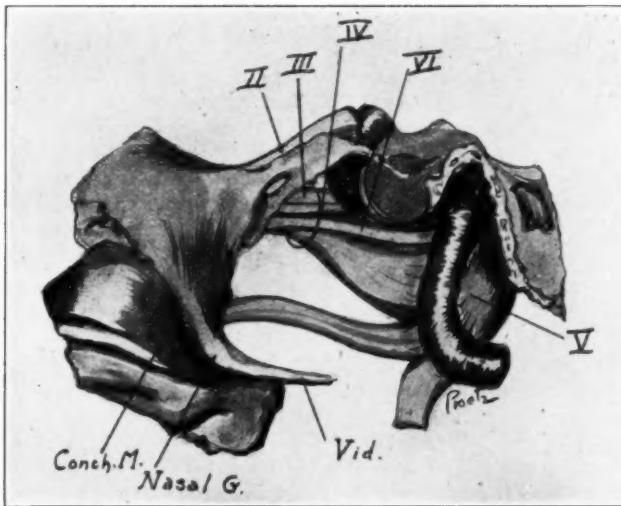
FIGURE 6.

The same dissection shown in Fig. 5, but viewed from the mesial side. The relations of the concha medialis, the nasal ganglion and the contents of the sphenoid fissure are shown.
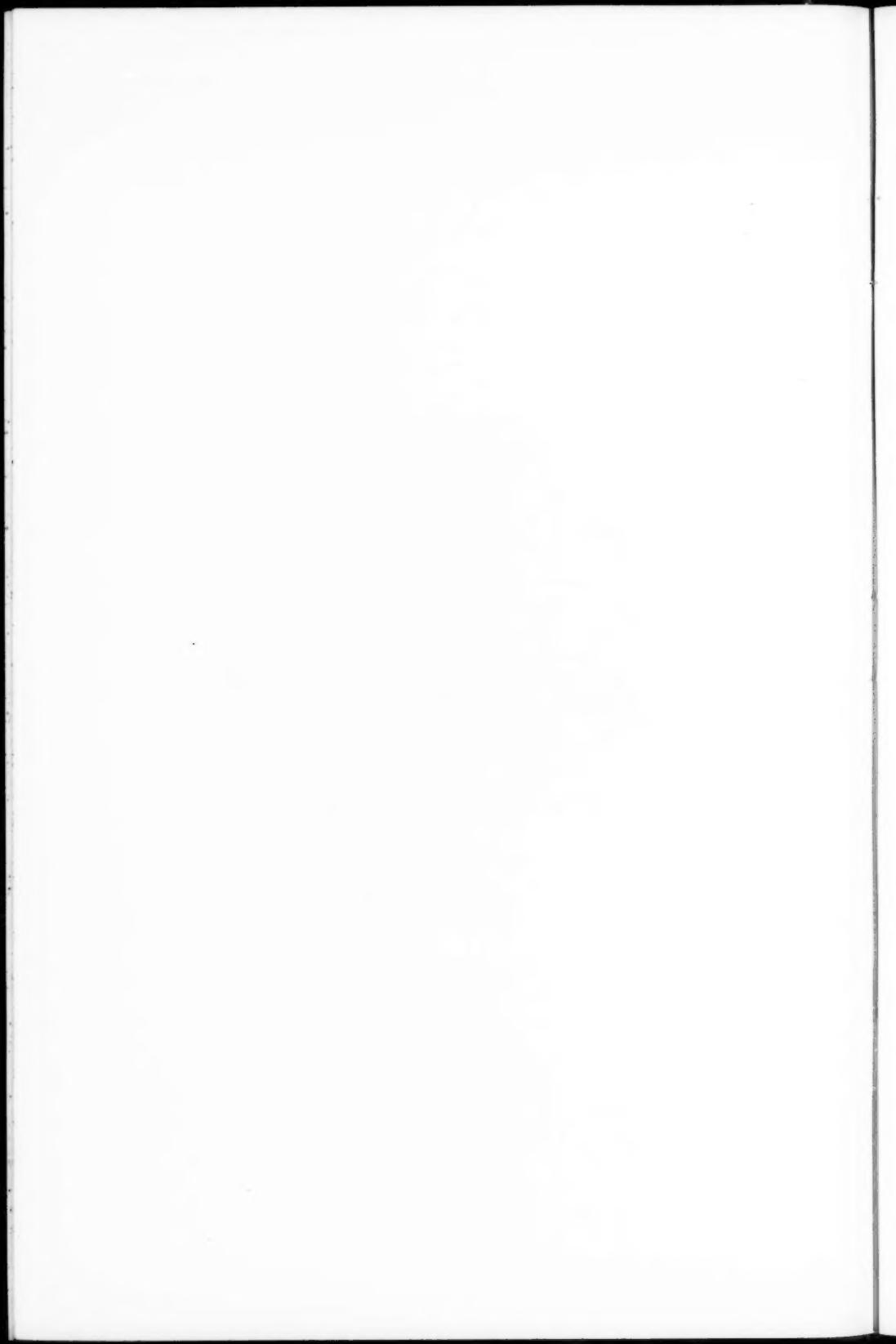
LXXVI.

DEAFNESS AS A SEQUELA TO MUMPS.*

By George H. Willcutt, M. D.,

San Francisco.

A number of articles have appeared on the subject of deafness as a complication or sequelæ of mumps during the last five years, the most instructive of same having been presented by Stern, Radin, Heilskov, Kaunitz and Albright. In the year 1860, Toynbee reported the first case of deafness associated with this disease and then followed reports of a number of other cases during a period of years from 1880 to 1890. In the year 1885, Pierce reported in the Manchester Chronicle an interesting series of forty cases which he had collected. However, it has remained for the past thirty or forty years a most unsettled pathologic question as to what is the anatomic cause of this severe, afebrile, incurable disease of the auditory nerve, which arises during or following acute epidemic parotitis.

To briefly summarize the various pathologic divisions or classes in which the reported cases have been placed, there are three main divisions: (1) those in which only the cochlear branch of the auditory nerve is affected, (2) those in which the vestibular branch is the only affected part, and (3) those in which both cochlear and vestibular branches are involved.

Varying theories of the mode of infection of the auditory nerve have also been expounded by the various authors, such as (1) direct extension, (2) direct transplantation, (3) by way of nerve sheaths or blood vessels, (4) by an acute exudation or metastasis in the labyrinth, and (5) a neuritis of meningitic origin. The authors of the last few years mentioned above have divided about as follows: Heilskov and Kaunitz favor the neuritis of meningitic origin, Albright and Weinstein and others, that of metastasis or exudative labyrinthitis. In brief, the favored theories are one or the other of the follow-

ing : a neuritis descending from the meninges toward the laby-
rinth, or a neuritis ascending from the labyrinth toward the
brain center.

With these two theories before him, the author of this work
has attempted to add something to the literature which may
prove of some benefit in the future work on the subject and
aid in clearing up the differences of opinion now existing. It
is with this hope that the following cases are reported in detail
and the suggestions offered for further investigations.

Since June 26, 1916, five cases of complete deafness follow-
ing or during an attack of acute epidemic parotitis have been
under the author's observation. In this series of cases, three
were in children between the ages of five and eleven years, and
two were female adults, aged 25 and 35 years.

Case 1.—In June, 1916, a woman telephone operator, 29
years of age, was referred with the following history: On
May 27th, she had contracted mumps from her young daugh-
ter, while nursing her through an acute attack of the disease,
and had been herself under the care of her family physician.
On June 2nd, six days after the onset of the attack, she suf-
fered greatly from vertigo, nausea and vomiting. This attack
gradually subsided during the following two days, and on
June 5th, she arose from her bed occasionally, but was unable
to walk without staggering, requiring assistance in moving
about the room. This condition of disturbed equilibrium per-
sisted for about seven days without other symptoms referable
to the ears. On June 15th, when attempting to use the tele-
phone, she noticed for the first time what she described as a
"peculiar feeling in the left ear," and then discovered she
was unable to hear conversation with that left ear. One week
later she presented herself for examination, and the hearing
tests showed a complete unilateral deafness of the left ear,
all tests being referred to the normal right ear. Caloric tests
showed a nonfunctioning left labyrinth. Although it was less
than a month from the onset of her labyrinthine symptoms no
spontaneous nystagmus was present, and compensation was
fairly well established. Treatments of some two months' dura-
tion with iodids and pilocarpin were unavailing. When seen
in October, 1916, the nonfunctioning labyrinth, both static and
acoustic, persisted on the left.

Case 2.—A little girl, aged 9 years, developed mumps on February 21, 1918. After an illness of four days, she suddenly had a "terrible noise" in her head, which lasted some minutes and stopped as suddenly as it began. The mother soon noticed that the child did not answer when spoken to concerning her condition, and the child then complained of being unable to hear anything. Examination that same night, February 25th, showed a complete bilateral deafness present but no clinical symptoms of labyrinthine irritation or destruction. At the same time the child complained of her eyes and of headache, stating that "everything seemed blurred and funny looking." No apparent involvement of the eye muscles or pupillary changes were noticeable, and on examination of the fundi the following morning showed only a slight inflammation of the nerve head and some congestion of the disc on the left side, with no abnormality in the right eye. At no time during this attack did the child complain of any vertigo or nausea. Some three months later, May 15, 1918, this patient was seen again and tested in the turning chair and by the caloric method, and normal· reactions were obtained from all semicircular canals, showing no involvement of the vestibular branch but a complete paralysis of both cochlear branches still present. A second eye examination at this time was reported as normal. Deafness persisted despite intensive treatment.

Case 3.—A young boy, aged 5 years, of Italian parentage, had been taken to the Pediatric Clinic at Stanford University Clinic in July, 1919, and a diagnosis of acute epidemic parotitis made there, the disease affecting the left gland only at that time. The usual home treatment was carried out by the mother, a very intelligent type, and the attack subsided after ten days. On the sixteenth day after the beginning of the trouble the boy complained of "some trouble like noise" in his head, not referable to either ear but causing him a great deal of annoyance. When put to bed that night he still complained of the head noise but soon fell asleep. The following morning, when awakened for some medicine, he put his hand to his right ear and told his mother he could not hear. The mother washed out his ears, removed a small amount of cerumen, and dismissed the matter for the time. She noticed repeatedly

during the following week that the child did not seem happy and kept putting his hand to his right ear. The case was then referred to me by a colleague, and examination on July 24, 1919, showed a complete unilateral deafness of the right ear, with slightly impaired hearing in the left. No symptoms of labyrinthine involvement were present nor had the child previously complained of vertigo. Another symptom of much interest and importance, was a hyperosmia, a hypersensibility of the olfactory sense. Young as he was, the child could detect the faintest odors and certain of these were objectionable to him. This condition persisted some weeks, gradually wearing off until normal again. No labyrinthine tests were obtained in this case, the patient leaving for parts unknown without notice. No eye symptoms present.

Case 4.—A young school girl, aged 11 years, very bright and standing high in her class work, was stricken with acute epidemic parotitis with involvement of both glands, in November, 1919. On the third day of her illness, she complained of buzzing in both ears and also of some pain as well. This pain was of the neuralgic type, being intermittent and of very short duration, radiating from the ears to the back of her head in the occipital region. Heat was applied by her attending physician with some relief. The buzzing continued, however, and when seen on November 20, 1919, the ear examination was then negative and the hearing normal in both ears. Despite due precautions, two days later, the patient stated she could not hear very well while lying with her right ear in the pillow. A second examination on November 24 showed a very different result from that made four days previously. A complete unilateral deafness of the left ear existed, the right ear being normal. During the four-day interval between examinations, two other important symptoms occurred, headache of basilar type and blurred vision. Vestibular tests showed normal functioning labyrinths on both sides.

Case 5.—A female adult, married, aged 35 years, was taken ill on October 15, 1921, and a diagnosis of mumps made by her attending physician. The disease began in the right gland and twelve hours later involved the left one as well. The right parotid was more swollen and painful than the left. On October 22, one week after the onset of the attack, the

patient noticed a ringing in the right ear, and some ten hours later some difficulty in hearing when using the phone, which stood on the right side of the bed, necessitating the use of the right ear. On the following day, the patient was suddenly seized with severe vertigo and nausea and she was unable to sit up in bed or stand on her feet without falling to the right side. This condition was greatly relieved by assuming the horizontal position and closing the eyes, although she had no turning sensation at any time. When seen on October 24th, ten days following the onset of her attack, the right gland was still somewhat swollen and painful, the right ear was completely deaf, vertigo and nausea present when standing or attempting to walk, no spontaneous nystagmus present, no spontaneous pastpointing, and the tinnitis practically gone. The patient complained of severe headache and of another symptom (previously mentioned in Case 3), hyperosmia, her sense of smell being extremely keen. The vertigo gradually diminished and the patient was up on her feet and about again on November 2, 1921. This patient refused to permit the vestibular tests so same were not obtained. On January 5, 1922, and April 14, 1922, when re-examined, the right ear was still completely deaf and the patient still experienced some vertigo when dancing and when leaning over to put on her slippers. No eye symptoms were present except a marked myopia, which was present prior to her attack in October, 1921.

Condensing the important points of these five cases, we find: (1) three children and two adults, (2) four female and one male, (3) four unilateral and one bilateral deafness, (4) three without labyrinthine symptoms (vertigo, nausea, etc.) and two with such symptoms; (5) one with a permanent non-functioning labyrinth, two with normal labyrinthine reaction and two not tested, (6) four with tinnitis or head noises and one with "peculiar feeling" in the ears, (7) three with definite headache and one with pain radiating to the base of the cranium, (8) two with hyperosmia and one without (two cases having been seen before the symptom was noticed) and (9) two cases had definite temporary eye symptoms and two without same (Case 1 not examined for same).

In reviewing the literature on this subject, I have failed to find any mention made of the last three symptoms given above.

namely, headache, hyperosmia and those referable to the optic
nerve. It may, therefore, be of some advantage to consider
these in connection with the auditory lesion, when attempting
to determine what anatomic condition is responsible for the
deafness found.

Among the various complications of mumps which have
been reported, that of meningitis has been mentioned as oc-
curring but has been considered as one of the less frequent.
The question that comes to our mind is, "What type of men-
ingitis do we have as a complication to mumps? What is the
organism causing it?" If we can have a meningitis due to
the pneumococcus, meningococcus, streptococcus, etc., why is
it not possible to have a meningitis due to the infectious organ-
ism of mumps, whatever that causative organism may be? A
mild meningitis of serous variety or type would have the
symptoms—abrupt onset, headache, nausea and vomiting, ver-
tigo, hyperesthesia, ataxia, pareses, strabismus, disturbances
of vision, disturbances of hearing, etc., or any combination of
these. If the meningitis be of the more severe type we would
look for convulsions, coma, opisthotonus, etc.

If we take the symptoms found in the cases reported in
this paper and consider them in the light of possible meningeal
symptoms, we can explain far more easily the involvement of
the various cranial nerves than to accept the theory that the
lesion is purely metastatic and affects each individual nerve
separately. In our studies of other cranial conditions, such as
luetic infection, we know that the auditory is the most vulner-
able of the cranial nerves and would therefore be the first in-
volved in any condition affecting these nerves. In a serous
meningitis, the group of nerves lying so close in the region
of the base would undoubtedly be the first ones affected by
such a process, and the more resistant ones would ultimately
recover while the more susceptible auditory nerve would be
permanently destroyed, in part or in whole.

It is with regret that this possibility of a meningeal origin
did not occur to me while the reported cases were under my
observation, as much more valuable data might have been ob-
tained at that time, but the desire to bring this matter before
those interested in this work with the urgent plea for further
investigation in an attempt to prove this meningeal origin,

causes me to offer the following suggestions: (1) An early spinal puncture and laboratory examination in cases of mumps with ear symptoms, headache, etc.; (2) a careful fundus examination and record of the visual field; (3) careful examination for an involvement of the other cranial nerves, especially the motor oculi, olfactory, facial and abducens. The condition is not as uncommon as thought, and much valuable information can be obtained if those seeing these cases will act promptly along the suggested lines.

516 SUTTER STREET.

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LXXVII.

CONGENITAL ATRESIA OF THE POSTNASAL ORIFICES.

By James E. Lebensohn, M. S., M. D.,

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Congenital occlusion of the choanæ is rare, but of extreme interest because correct diagnosis may often be a matter of fundamental importance. Richardson[1] considers this as one of the most frequent types of congenital malformation of the nasal chambers. He states: "Many of this class of cases are unrecognized at birth, and when speedily dying as a result of the obstruction to respiration are placed under the general class of asphyxia neonatorum. The number of observed cases has increased greatly in the past twenty years, a tribute to the increased number of intelligent workers in rhinology; and to the knowledge more widely diffused that such a deformity may occur."

Among children, many of these cases come to the physician with a history of nasal obstruction, and are diagnosticated "adenoids." Kirby[2] reports two cases in both of which tonsils and adenoids had been removed and later secondary adenoidectomy had been done.

RETROSPECT.

The first recorded case appeared in 1830 and concerned a fetus (Otto). In 1854, an operation was reported on the first case noted in the living (Emmert). The total number of cases in the literature in 1886 was 17 (Hubbell); in 1898, 21 (Clark); in 1904, 61 (Schmiegelow); in 1910, 115 (Fraser). Since 1910, I find in the archives record of 54 cases. The present total, inclusive of the case I now report, is thus 170. Of special interest to the obstetrician and the pediatrician are those cases that have been observed shortly after birth.[3,4,5,6,7]

PATHOLOGY.

Congenital choanal atresia has been reported three times as often on both sides as on one side alone, and the obstruction

when present is usually complete.   In 90 per cent of the cases the occlusion has been of the osseous type.   The obstructing plate of bone is vertically placed, has a concave pharyngeal aspect, and is situated within the nasal chamber, the posterior edge of the vomer extending about a millimeter beyond it.

The central portion of the occlusion is thinner than the periphery and may sometimes be composed of membrane only. In this case postnasal examination generally shows a more or less marked dimpling at this point (Fig. 1)—occasionally a perforation.   However, this opening, when present, is functionally of little value, as an aperture the size of a crow's quill gives the patient neither the ability to blow the nose nor to respire.

The accepted embryologic explanation of the typical form of congenital choanal atresia just described is this: The primitive nasal cavities commence as blind cul-de-sacs.   These cavities elongate, and finally the obstructing nasobuccal membrane gives way.   In case of choanal atresia, however, this membrane has become permeated by mesoderm and so fails to break down.

CHOANAL OCCLUSION IN THE NEWBORN.

The symptom complex of choanal occlusion in the newborn is quite different from that in the adult.   In the newborn, bilateral occlusion of the choanæ constitutes a definite emergency, and the life of the infant may depend upon making the diagnosis.   The distinctive symptom is "cyclic dyspnea" (Richardson).[4]   The child struggles for air, the face becomes slightly cyanosed, the child cries, and the mouth breathing instituted relieves the distress.   The child rests, and the cycle of respiratory difficulty recurs.   So great is the dyspnea as to prevent the infant from taking either the breast or the bottle. Since mouth breathing is an acquired habit, the newborn child thus affected suffocates unless special measures are taken.

Thick, gelatinous, clear mucus fills the nasal cavities—another characteristic, pathognomonic of this affection.

To verify the diagnosis:

1. Syringe the cavities.   The fluid does not enter the pharynx but returns in excess at the anterior nares.

2. Pass a probe. The resistance of the obstructing osseous plate is felt.

In the management of this emergency, the immediate object is to secure and maintain mouth breathing. By pressing the lower lip down the dyspnea is relieved. The lips are thus kept apart, day and night, by a relief of nurses, until the end of the second week, by which time the child has probably learned to breathe through its mouth. During this period nutrition is maintained by dropper feeding. The child shortly learns to breathe through its mouth and suck at the bottle alternately.

After the ninth month the nasophraynx is sufficiently well developed to permit of the finger manipulation necessary to operation. To prevent possible alterations in the facial skeleton the operation should be undertaken at this period or soon thereafter.

CHOANAL OCCLUSION IN LATER YEARS.

Choanal atresia in later years likewise produces a very definite clinical picture. On the side of the choanal closure the following constant phenomena are observed:

1. Nasal obstruction of the completest type. Neither respiration nor expulsion of mucus is possible. In crying, the tears flow out of the anterior nares, owing to their inability to pass into the pharynx.

2. Anosmia occurs because of the absence of an air current. After removal of the obstruction the sense of smell is sometimes partially regained.

3. Accumulation of a distinctive type of mucus, unknown in any other nasal condition. The mucus is thick, albuminous, extraordinarily tenacious, and of a peculiar bluish color. Because of retention it is sometimes offensive, and by irritation may occasion a regional dermatitis. The patient, by pressure against the alæ, may be able to remove the viscid secretion.

4. Atrophy of the turbinals, makes the nasal cavity appear more spacious than usual. The posterior tip of the inferior turbinate may be wanting. The mucosa is very pale, sodden and inelastic; and the inferior turbinate may show mulberry degeneration (Fig. 2). The atrophy is due to lack of use and to the pressure of the masses of mucus.

5. Deviation of the septum toward the obstructed side in unilateral cases. Several causes combine to produce this effect:

(a) The occlusion holds the septum to its side (well shown in X-ray, Fig. 3).

(b) The obstructed side is not subject to the dilating influence of the air current.

(c) The V-shaped groove in the upper border of the vomer may be maldeveloped on this side, so that the cartilage has slipped out and projects under the mucous membrane.

6. History of difficulty in maintaining nutrition in infancy. Among variable symptoms that may or may not be present are: Other anomalies of developmental arrest; mental deficiency; nasal voice; moderately separated lips;—in bilateral cases, auditory involvement and disturbance of taste;—in unilateral cases, facial asymmetry, unilateral sweating and inequality of the palatal arch (the arch being higher and narrower on the side of the choanal occlusion).

THE OPERATION.

The usual technic has been to operate under general anesthesia. Experience with the local anesthetics in this operation has not been satisfactory. With the finger in the pharynx as a guide, the occluding bone is removed by the electric burr or with chisel, punch or curette. Special care is taken to remove entirely the projecting border of the vomer and to smooth down nicely the edges of this newly made aperture, for otherwise recurrence of the closure by granulations will take place. The operation is easily done and without much hemorrhage.

In the after treatment, Thomson[5] relies on simple syringing, but I have not found this sufficient to keep down the granulations. I would recommend the method of Brady.[6] He finishes the operation by passing around the septum a strip of bismuth impregnated gauze, and tying this around the columella. This is changed daily by attaching a fresh strip of bismuth impregnated gauze to one end of the old strip and then pulling on the free end. The change is painless and is kept up for ten days.

Another method for preventing the growth of occluding granulations from the vomerine border is to combine the re-

moval of the obstruction with a thoroughgoing submucous resection of the septum.[10] This procedure is all the more justified, as a marked deviation of the septum is always present in this condition.

The use of rubber tubes in the after treatment occasions an irritating discharge and is not recommended.

CASE REPORT.

The patient—an electrical worker, white male, 23 years old, of good muscular development, weighing 144 pounds, and 5 feet 7½ inches tall—was admitted to the U. S. Veterans' Hospital, Maywood, Ill., July 19, 1922, for weakness in the right leg. The diagnosis was "thrombophlebitis of the right femoral vein following pneumonia contracted while in the U. S. Naval Service."

This man came under our observation in the routine otolaryngologic examination which every patient admitted to the hospital receives. He was phlegmatic in temperament, had a grammar school education, and was of average mentality. The patient volunteered no complaint of nasal trouble, but on questioning admitted difficulty of breathing on the left side. He had twice been "operated on for adenoids" without relief and had become resigned to his nasal condition.

Examination: The right side of the face is slightly fuller than the left (Fig. 5).

Anterior rhinoscopy reveals a normal right naris with the turbinals well developed and moderately turgescent.

The left naris is filled to a considerable extent with a clear tenacious mucus. Later examination showed that this mucus was most abundant in the morning and that by evening the patient had wiped away most of the secretion. The appearance of the turbinals in this naris was striking. The inferior turbinal showed an advanced degree of mulberry degeneration (Fig. 2). The mucosa was anemic and sodden. Owing to the undeveloped condition of the turbinate bodies, this nasal chamber appeared much roomier than the right (though it really was much smaller—note skiagram, Fig. 4). The septum was free from spurs and appeared to anterior rhinoscopy only slightly deviated to the left.

Postnasal examination was facilitated by a roomy and almost insensitive pharynx. Even a laryngeal mirror could be used and the case easily demonstrated to the most inexperienced nasal examiner. The right choana was without pathology. The left choana was distinctly smaller than the right, and about one millimeter external to the vomerine border was the occlusion shown in Fig. 5. In the center was an invagination that measured 1 mm. transversely, and 2½ mm. vertically. It appeared like a perforation, but syringing the nostril with methylen blue tinted fluid revealed no passage. A transilluminated view of the occlusion was secured with a Cameron diagnostic lamp in the left naris. The osteomembranous character suggested was subsequently confirmed by operation.

Testing the inspiratory and expiratory blasts demonstrated absolute blockage on the left side. Likewise anosmia on this side was complete.

The only other abnormality with the nose and throat was a bifid uvula, the left division of which was the longer. The palate was normally arched with the raphe in the midline.

Both ears were alike without pathologic process and responded with equal normality to all tests. In these unilateral occlusions it has been repeatedly observed that hearing is not affected. This finding discredits the idea that the common forms of intranasal obstruction, such as spurs, deviated septum, etc., are a determining factor in impaired hearing. However, they may be an indirect influence by localizing catarrhal processes to the eustachian tube of the affected side.

The skiagrams (Figs. 3 and 4) demonstrated clearly on the affected side the undeveloped nostril, the atrophied turbinals, the bony periphery of the occlusion and the marked posterior deviation of the septum. The accessory nasal sinuses were all clear.

Communication with the parents in regard to the early history of the patient elicited the following interesting statement: "As an infant he had a discharge from his nostrils and nursed with difficulty. When he was about two weeks old something green and about an inch long came out of his right nostril on sneezing. We thought the trouble with his nose was a cold and applied hot towels as our doctor instructed, but the left

nostril never became free." A photograph of the patient as an infant shows the face to be perfectly symmetrical (Fig. 6.).

The operation was performed under light ether anesthesia and completed in five minutes. Simply, the bony occlusion was removed with the posterior border of the vomer and the edges of the newly made aperture smoothed. No packing was introduced. The after treatment consisted only of cleansing sprays. The recovery was rapid. The patient could now breathe as freely through the left nostril as through the other. The sense of smell, too, was partially regained. Strong odors, such as turpentine, were now recognized, though not tobacco or more delicate scents.

A letter from the patient six months later informed me that a sense of obstruction was developing in the operated nostril and that the mucus, which was once more continually being formed, was becoming increasingly difficult to blow out.

I am hoping that I may have the opportunity to operate once more on this case. In this event, after excising the granulations that have formed, I shall remove the posterior part of the septum by submucous resection. This, I believe, should prevent a recurrence of the obstruction.

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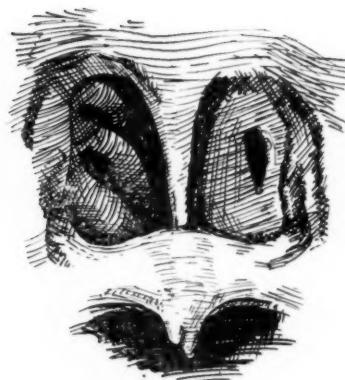
Fig. 1. Congenital occlusion of the left posterior choana. The black area in the center represents the membranous portion that appears dark because invaginated.

Fig. 2. Mulberry degeneration of the left inferior turbinate. The degeneration is probably due to the constant irritation of the accumulating mucus.
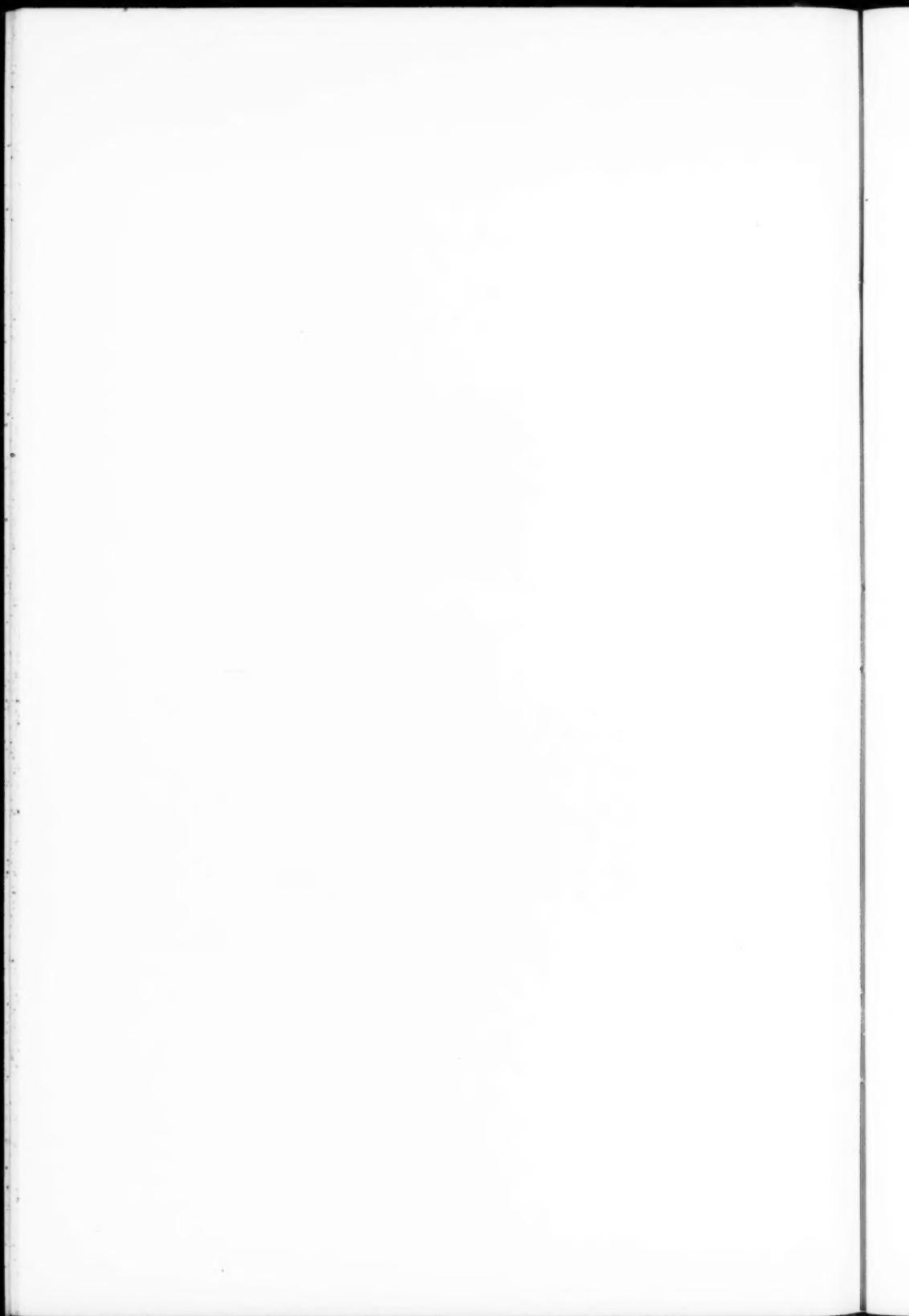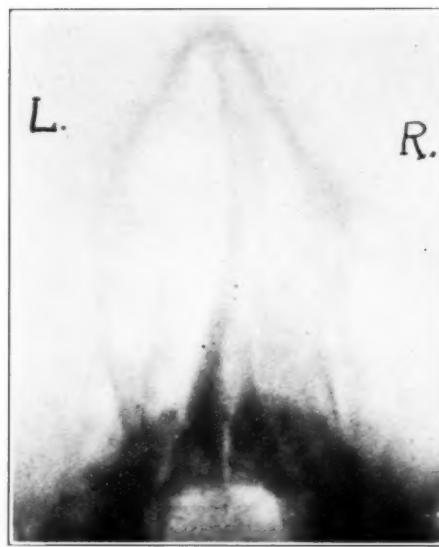
Fig. 3. Skiagram showing posterior deviation of the septum, and atrophied condition of turbinals in left naris. The small size of the left posterior choana is very well indicated.
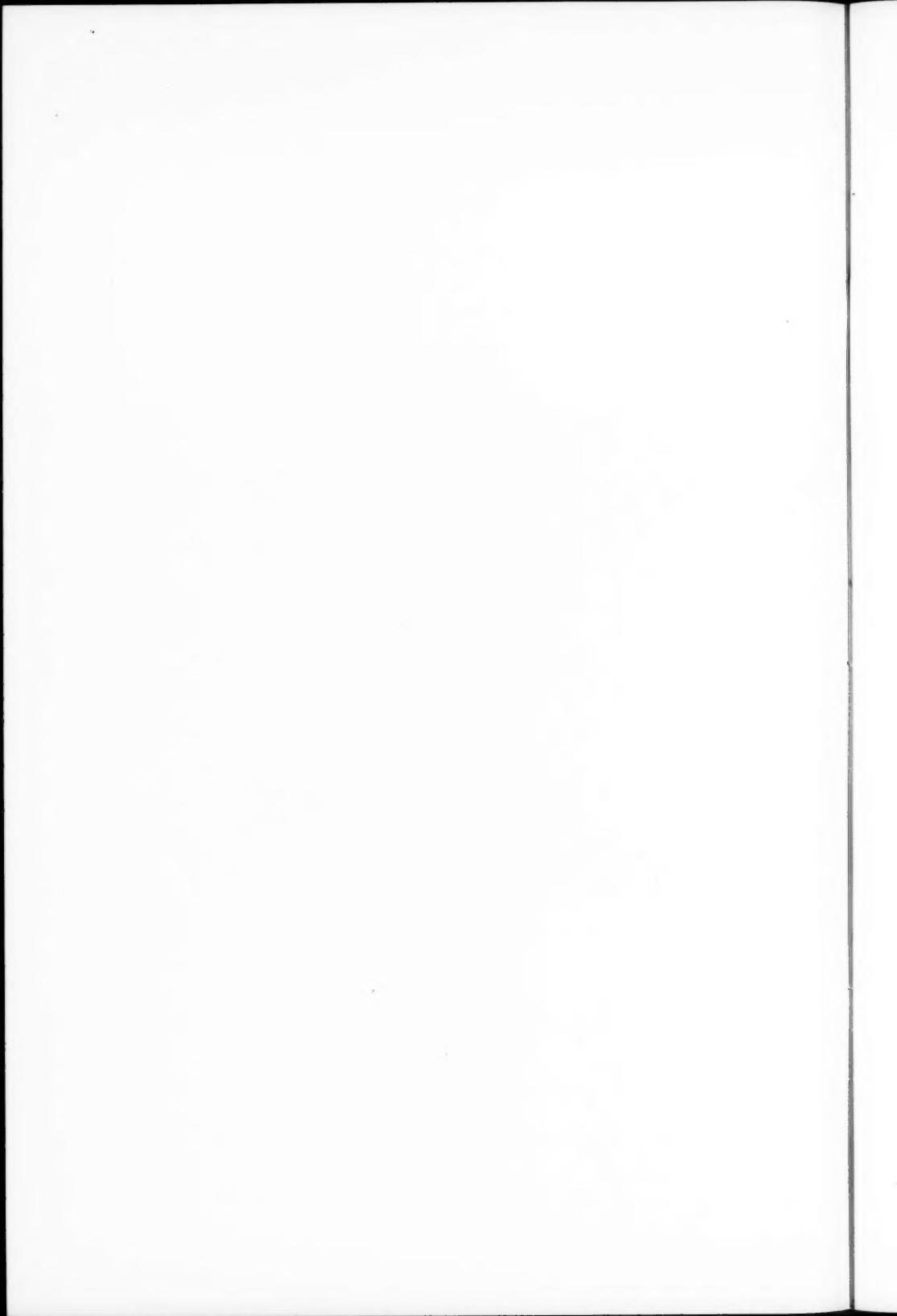
Fig. 4. Skiagram showing the undeveloped left naris with its undeveloped turbinates. The osteomembranous condition of the occlusion is better suggested in the original film.
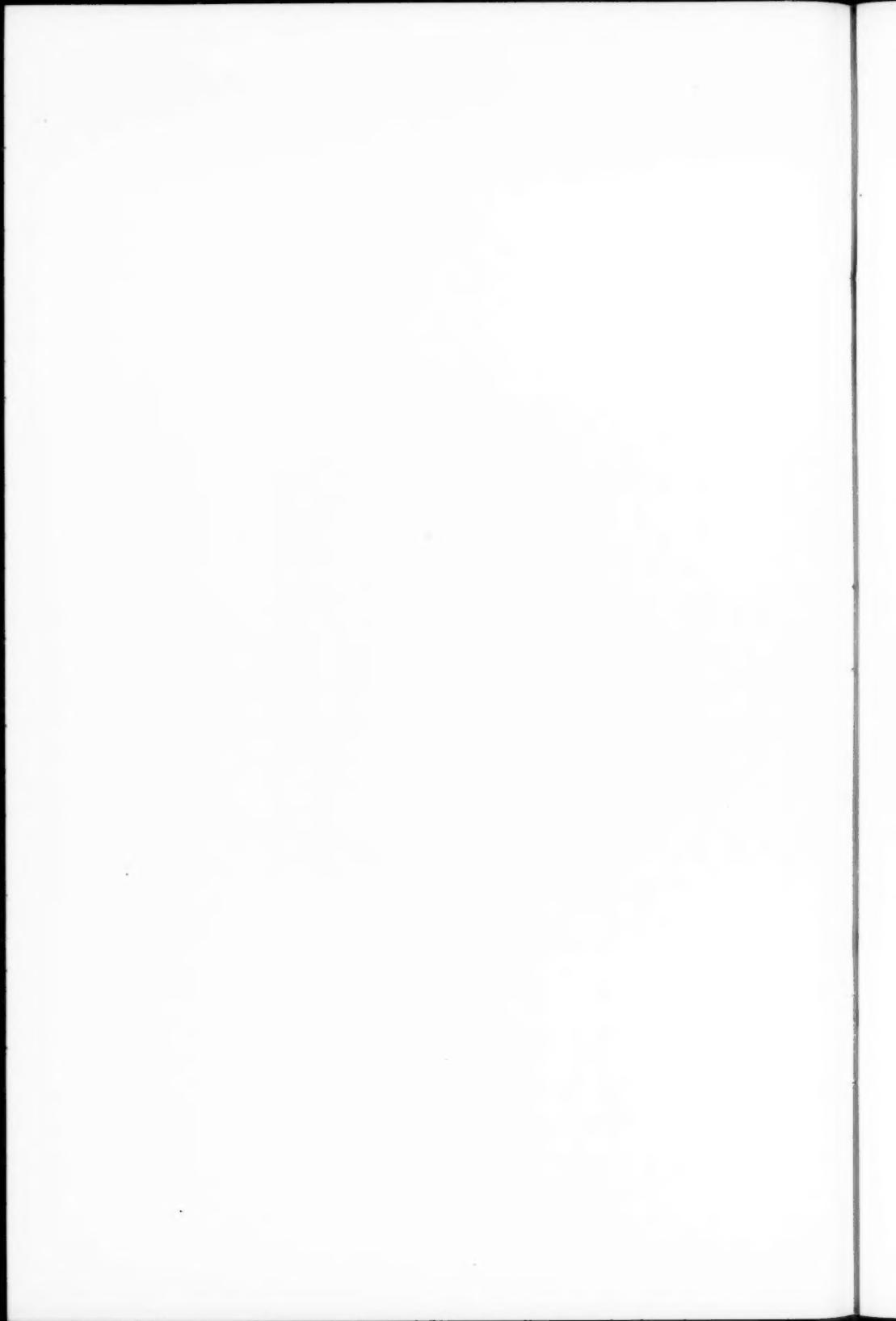
Fig. 5. The patient at age 19. The right side of the face is somewhat fuller than the left.
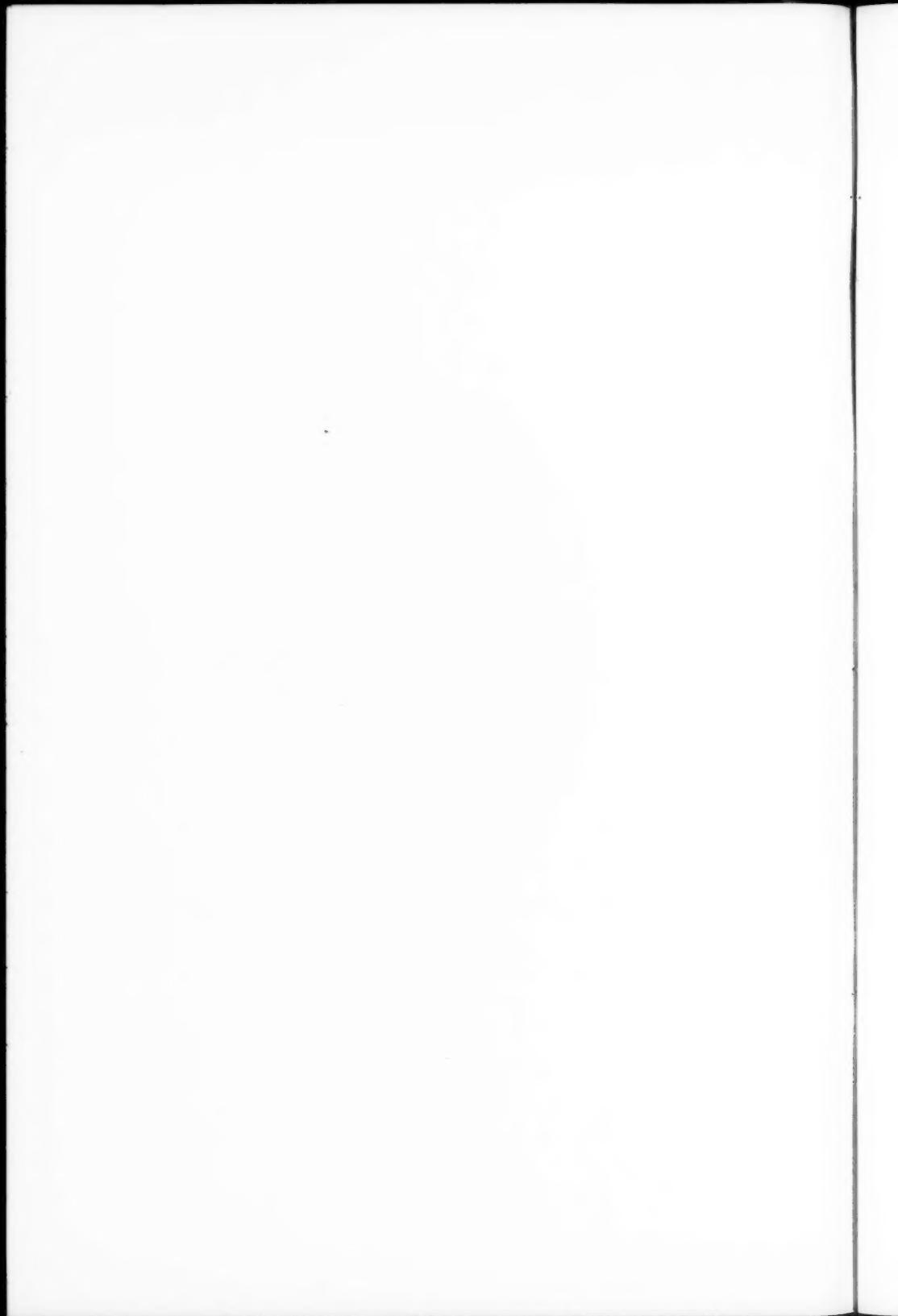
Fig. 6. The patient as an infant. The face appears entirely sym-metrical.
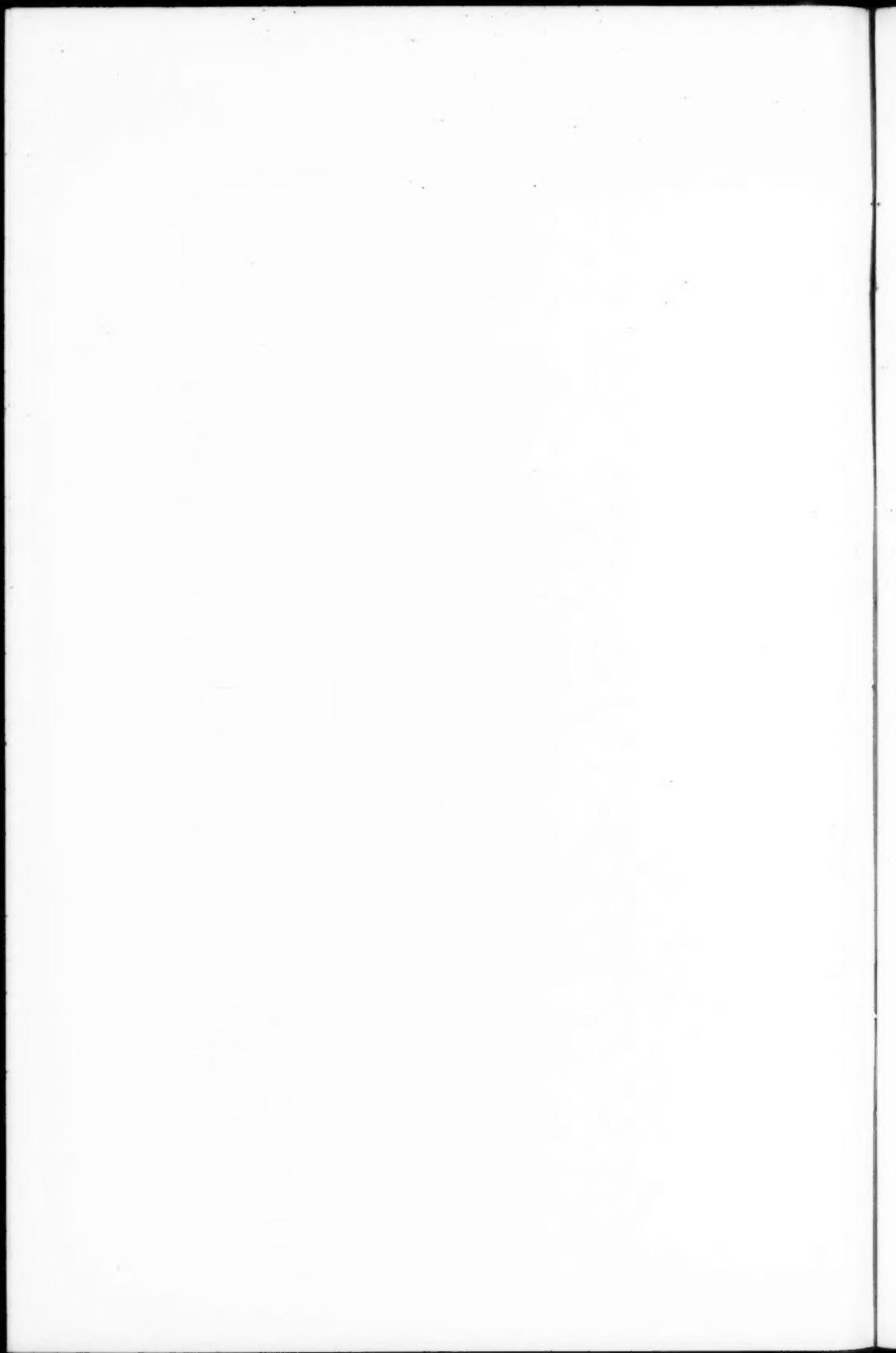
LXXVIII.

NONTRAUMATIC HERNIA OF THE DIAPHRAGM. AN EMBRYOLOGIC VIEWPOINT.*

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With the recent advances in roentgenologic diagnosis and the constant perfection of operative technic the problem of diaphragmatic hernia has become more and more a surgical one, resulting, in the nontraumatic cases, in the persistent neglect of their etiology, their commonest points of occurrence and their relation to the normal landmarks of the diaphragm. The confusion and uncertainty underlying this surgical enthusiasm is well shown by a quotation from one of the leading surgical journals. "Congenital hernias do not need to be present at birth; hernias should be considered congenital, when they are formed, because there is a weak point in the diaphragm. . . . This is generally situated around the esophagus. . . . We do no know if there are cases of hernia through the opening for the aorta or vena cava. . . . Diaphragmatic hernias differ from all others because . . . they are deprived of an enveloping sack." Another author states that "false hernias are always traumatic." These statements indicate not only uncertain but inaccurate knowledge of the fundamental principles of diaphragmatic hernia. Surgeons generally assume certain weak spots to be the sites of hernia, regardless of where these are or why they occur.

I have undertaken in this article to correlate the embryology of the diaphragm, so far as this is definitely understood, with the subject of nontraumatic hernia, explaining the frequency of the various types and their points of occurrence, in the light of what is known of the elements which compose the diaphragm. I have also collected all the nontraumatic

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*The investigation of this subject was made at the suggestion of Dr. Harris P. Mosher, Professor of Laryngology, Harvard Medical School.

cases reported since 1900 and tabulated those reported pre-
viously by Thoma and Grossa (Fig. 11 and tabulation).

ANATOMY.

The adult diaphragm is a musculofibrous septum arising
from the circumference of the thorax. It is elliptical in shape,
composed of a peripheral muscular portion and a central ten-
dinous portion. It is attached in front by fibers to the ensi-
form cartilage, on either side to the inner surface of the car-
tilages and bony portions of the six or seven inferior ribs,
interdigitating with the transversalis muscle, and behind to
two aponeurotic arches, the internal and external arcuate liga-
ments. Centrally it is attached to the lumbar vertebræ on each
side by its crura. There is a muscular deficiency on either side
of the slip from the ensiform cartilage, filled by areolar tissue
covered on the thoracic side by pleura and on the abdominal
side by peritoneum. These deficiencies form the socalled
foramina Morgagni or Larry's spaces. There are also simi-
lar triangular areas devoid of muscle posteriorly between the
fibers arising from the internal, and those arising from the ex-
ternal, arcuate ligaments known as the foramina Bochdalecki.

The central tendon of the diaphragm is a thin strong apo-
neurosis shaped like a trifoil situated immediately below the
pericardium, with which it is partly blended.

The openings connected with the diaphragm are three large
and several smaller apertures. The former comprise those
for the aorta, the esophagus and the vena cava. The aortic
opening is the lowest and most posterior of the three large
apertures, being on a level with the first lumbar vertebra. It
is slightly to the left of the midline, immediately in front of
the bodies of the vertebra and hence, strictly speaking, it is
not in the diaphragm at all but behind it, the anterior margin
being formed by a tendinous arch thrown across from the crus
on one side to that on the other. It transmits the aorta, the
vena azygos major and the thoracic duct. The azygos major
may pass through the right crus.

The esophageal opening is situated at the level of the tenth
dorsal vertebra. It is elliptical in form, muscular in structure
and formed by the decussation of fibres from the two crura.
It is anterior to, and a little to the left of, the aortic opening

and transmits the esophagus, the two vagi and small esophageal vessels.

The opening for the vena cava is on a level with the disc between the eighth and ninth dorsal vertebræ. It is quadrilateral in shape, lies entirely within the central tendon and transmits the inferior vena cava.

The right crus transmits the greater and lesser splanchnic nerves of the right side, the left crus those of the left side together with the vena azygos minor.

The diaphragm is covered above with the right and left pleura and the pericardium, below by the peritoneum. It is in relation on the right with the right lung and the liver, on the left with the left lung, stomach, spleen and left kidney.

The phrenic nerve, originating from the fourth and fifth branches of the cervical plexus, in conjunction with the fifth and sixth intercostals, supplies the diaphragm. The left phrenic nerve is longer than the right, since the left half of the diaphragm is lower than the right and because the heart protrudes on the left side.

COMPARATIVE ANATOMY.

The only animals showing a complete formation of a true diaphragm are the mammals. In the lower forms it is only the pericardial cavity which is separated from the abdominal, and there may even be a persistent pericardioperitoneal canal connecting them. In some urodeles there is found an analogous but not homologous muscular structure springing from the vertebral column and ribs which forms a partition between the pleural and peritoneal cavities but which is complete on both sides only in crocodiles. In this partition there is no evidence of a central tendon; and Keith alone upholds any homologous relation between the two structures.

EMBRYOLOGY.

In the early human embryo the pericardial, pleural and peritoneal cavities communicate, all three leading indirectly into the extra-embryonic coelom. At this time the pericardial cavity is a paired structure except anteriorly, where each side is connected with the other to form a U shaped cavity. In this cavity the heart develops and with further growth

the splanchnic mesodermal layers are folded together to
form the ventral wall of the pericardial cavity (Figs. 1 and 2),
being attached for a short time to the heart by the ventral
mesocardium, a structure which soon disappears to leave the
heart free at this point.   Passing forward toward the heart
on the splanchnic layer of the mesoderm covering the yolk
stalk are the large vitteline veins, one on either side, converg-
ing to an acute angle to form the sinus venosus. This angle
between the veins is filled with a solid mesodermal structure
forming the ventral mesentery and first called by His the
septum transversum. This structure forms the earliest anlage
of the future diaphragm (Fig. 3). The vitelline veins soon
become so large and expand so far laterally that they bring the
splanchnic mesoderm in which they lie in contact with the
somatic mesoderm which forms the lateral wall of the peri-
cardial coelom (Fig. 4). Fusion of the two layers of meso-
derm along the course of the veins now takes place, and each
lateral coelomic or parietal recess becomes divided into two
parallel passages, a dorsal and a ventral parietal recess (Fig.
4). The angle at which the veins unite and their lateral
divergence result in the closure caudally of the ventral re-
cesses and in the interruption of their continuity with the
trunk cavity, so that they form two blind pouches extending
a short distance below the mesodermal mass in which the
juncture of the veins lies and which forms the floor of the
pericardial cavity. The dorsal recesses, on the other hand,
retain their connection with the trunk cavity by way of the
socalled pleuropericardial canals for a much longer period.

This fusion of splanchnic and somatic mesoderm laterally,
together with the transverse septum, forms a thick semilumar
mass which projects horizontally into the coelom with its free
edge facing dorsally. Laterally it is continuous with the body
wall, mesially it is continuous with the dorsal mesentery, a
structure which connects the pericardium with the vertebral
column and which contains the foregut. The latter is con-
· nected by the ventral mesentery to the anterior body wall, and
into this mesentery, caudal to the septum transversum, grows
the liver. This structure pushes into the substance of the
septum, which thus acquires a considerable thickness, espe-
cially toward its dorsal edge (Fig. 5A). It furthermore be-

comes differentiated into two layers, an upper and thinner, which form the floor of the ventral portion of the pericardial cavity, and a lower and thicker, which contains the liver. In later stages the layer containing the liver becomes separated from the upper by grooves or infoldings of peritoneum, which gradually deepen medially and dorsally, but without actually reaching the midline, a portion of the septum remaining as a midline fold or the adult falciform ligament, the free edge of which contains the umbilical vein (Fig. 5B). The failure of these grooves to separate the liver completely dorsally gives rise to the permanent coronary or falciform ligament.

Thus there is formed a ventral separation of the pericardial and peritoneal cavities. A dorsal communication still exists between them through two narrow, short canals, which later give rise to the pleural canals or cavities. The further separation of the pericardial cavity from the two pleural cavities, and of these in turn from the peritoneal cavity, is an extremely complicated process and one concerning which embryologists differ, but it is the closure of this portion of the communication with which we are most concerned in hernia of the diaphragm. The study of this procedure is rendered all the more difficult by the fact that there is a constant increase in the flexion of the embryo, such that the pericardial cavity comes to assume a ventral instead of a cephalic relation, thus making serial sections confusing. The chief factors in the further closure of the dorsal canals are the Cuverian ducts. They are the result of the union of the anterior and posterior cardinal veins developing in the lateral body wall to form the common cardinals which gain entrance to the heart through the fusion of the splanchnic and somatic mesoderm described above. The protrusion of the ducts toward the dorsal coelomic cavities causes, first of all, a diminution in the size of the latter and then gives rise to a circular ridge, called by Mall the pulmonary ridge, extending from below on the dorsal wall near the top of the Wolffian body, upward, laterally, anteriorly, and then caudally to the free edge of the transverse septum. This ridge later broadens and its edges become thicker and more prominent than its central portion in such a way that a triangular protrusion is formed, the cranial and caudal margins forming the sides,

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the dorsal body mesentery the base of the triangle (Fig. 6). The cranial margin or side of this protrusion forms the pleuro-pericardial membrane, and its further extension and growth on each side reduce the communication between the pericardial and pleural cavities to a mere slit. The caudal margin becomes the pleuroperitoneal membrane and eventually separates these cavities.

In two-millimeter embryos the septum transversum lies about opposite the middle cervical region, in a transverse position. According to Mall, there is a gradual migration caudally until at 24 mm. it is opposite the first lumbar segment. It is at the time when it is opposite the fifth cervical segment that the septum receives the phrenic nerve. With this descent there is a reversal of position almost at right angles to its earlier one, so that the original dorsal surface becomes ventral. This descent also serves to carry the pleuroperitoneal membrane caudally, in such a way that the lung, which is meanwhile developing from the walls of the dorsal mesentery, comes to lie in the triangular space between the pleuro-pericardial and pleuroperitoneal membranes (Fig. 7). The latter is still unclosed, and for a time the lung projects below it into the abdominal cavity. With the descent of the septum the dorsal end of the pleuropericardial membrane lags behind and thus comes to assume a position nearly at right angles to the septum. Since the communication between the pericardial cavity and the pleuroperitoneal canals is greatly narrowed by a general expansion of the peripheral tissue—i. e., laterally by the pulmonary ridges, medially by a thickening of the dorsal mesentery to form what Uskow calls the longitudinal mass, it is a simple matter for the pleuropericardial membranes to fuse with this longitudinal mass and thus cut off the pericardium completely. It is not till somewhat later that the pleuroperitoneal membrane fuses with the dorsal mesentery, thus separating the peritoneal cavity from the pleural cavities. The lungs have by this time receded and the septum descended sufficiently to include them within the pleural cavities.

The lungs begin to develop in the longitudinal mass on the medial walls of the pleuroperitoneal canals, into which they bulge laterally. The right lung develops further down in the

pleuroperitoneal canal than the left.   At the time of closure of the pleuroperitoneal openings the membrane accomplishing this is so placed that its lateral half extends much further cephalic than its medial half, so that a transverse section through it would show peritoneal cavity external to and above the pleural cavity, a relation exactly opposite to that in the adult.   This early relation is reversed by the caudal growth of the lungs, which push the diaphragm laterally until a section shows the pleural cavity laterally and the peritoneal medially.

Growth of the lungs laterally is responsible for the further development of the true pericardium, which is at first closely bounded on each side by mesoderm of the body wall of which it is really a part (Fig. 8A).   Later the lungs push forward around the pericardium, splitting off a thin layer which forms the real pericardial sack covered laterally with parietal pleura (Fig. 8B). The phrenic nerve first grows between the subclavian veins and cardinal veins along the lateral wall of the pleural coelom.   With the subsequent separation of the pericardial from the pleural coelom by the growth of the pleuropericardial membrane, the nerve is left on the lateral edge of this separating membrane.   Later when the lung burrows to extend around the side and front of the heart the nerve is pushed into the pericardial wall between the heart and the lung.

The above description is concerned only with the membranous separation of the three body cavities.   The adult diaphragm is largely a muscular structure, the full development of which takes place much later.   The muscle elements invade the diaphragm while it is still high in the neck, originating mostly, according to Sobotta, from the fourth cervical myomere.   The muscle grows in from the lateral body walls to a certain point, after which its further centripetal extension is accomplished by pressure from the expanding lungs, which peel off the muscle along the body wall as they advance downward.   In early fetal life the entire diaphragm, like the septum of some animals, is composed of muscle, and the tendinous portion originates only secondarily by an atrophy of the central part of the muscle.

Another structure of embryologic significance in the study of diaphragmatic hernia is the superior omental recess. This is a cranial projection of the general abdominal cavity, at first bilateral, on either side of the dorsal mesentery of the gut. This fingerlike extension on the left side is obliterated at an early stage in development, but that on the right side persists for some time and eventually gives rise to the superior omental bursa (Fig. 9). At one stage in its development its extreme upper or cephalic portion becomes narrowed and cut off at the level of the esophagogastric union to form a blind pouch. Normally this either disappears by obliteration or persists as a small bursal sack enclosing the esophagus on all sides except at its mesenteric attachment posteriorly, just as the testicle is enclosed in the tunica vaginalis. In rabbits this bursa, called the infracardiac bursa, is constant; in adults it is usually not evident. It may be entered by splitting the leaves of the ligamentum pulmonale, just below which it lies, whereby a sound can be passed directly through the diaphragm into the abdominal cavity and lesser sack as in a congenital inguinal hernia.

Thus the adult diaphragm is derived from a number of separate elements which, in the order of their appearance, are as follows (Fig. 10) :

1. Septum transversum (ventral mesentery). Gives rise to the ventral and central portion of the diaphragm, especially the part directly below the heart.

2. Dorsal mesentery. Gives rise to the posterior and central part containing the esophagus.

3. Pleuroperitoneal membrane. Gives rise to the posterolateral portion of the diaphragm where the costal and lumbar segments meet.

4. Septum transversum (lateral extensions). Gives rise to costal portion on each side.

5. Thoracic wall (peripheral part, especially laterally).

It is a difficult matter to obtain a clear mental picture of the processes involved in the union of these varied elements, the more so since they take place in all three planes at a time when the embryo itself is constantly changing its shape and relations. A helpful if not entirely accurate simile is that suggested by Waldeyer,[1] in which he describes the separation of

the coelomic cavities in terms of the nasopharynx, an ana-
tomic field which is much more readily visualized than the
developing body cavities.   He conceives of the anterior nares
as closed and the anterior portion of the nasal septum absent.
This single cavity then represents the pericardium.   Prolong
the septum backward till it crosses the nasopharynx and
touches the posterior pharyngeal wall, thus dividing the
pharynx into two parts representing the two pleural cavities
which become united below the lower edge of the septum with
the esophagus, the latter representing the peritoneal cavity.
Approximate the tongue to the hard and soft palate, the tongue
representing the liver, the hard palate the septum transversum
and lateral mesocardium, or together, the ventral component
of the definitive diaphragm.   The soft palate is the pleuro-
pericardial membrane which, passing from the posterior part
of the ventral diaphragm, finally fuses with the posterior phar-
yngeal wall to separate the nasal cavity from the two pharyn-
geal or pleural cavities.   The tonsillar pillars or pulmonary
ridges are to be conceived of as originating below on the pos-
terior pharyngeal wall and as passing upward and forward in
a concave arch to fuse with the "dorsal diaphragm" or soft
palate, forming a crescentic lateral ring or groove from
above downward and backward.   Thus a triangular shaped
area is outlined by the soft palate and pillars, the gradual nar-
rowing and constriction of which, together with the lateral
thickening of the septal mass, closes off each half of the
pharynx from the esophagus or each pleural cavity from that
of the abdomen.

The foregoing account of the embryologic development of
the diaphragm is in accordance with the views accepted by
the majority of embryologists.   Keith,[2] however, has a quite
different concept, based on, and supported by, analogies from
the point of view of comparative anatomy.   According to him,
the pleural cavities were never a part of the general coelomic
cavity but were developed in the body wall by extrusion of the
lungs, much as the testicles are extruded from the abdominal
cavity, retroperitoneally, the internal abdominal ring corre-
sponding to the pleuroperitoneal opening.   He found that in
amphibians the lungs were in the abdomen below a diaphragm
composed of the transverse septum (analogue of the mamma-

lian ventral diaphragm), and a fascial layer of cervical apo-
neurosis.   This diaphragm is supplied with muscle derived
anteriorly from the rectus, posteriorly from the cervical por-
tion of the transversalis.   In reptiles this "abdominal" lung
pushes up and evaginates the whole dorsal fascia over it.   In
birds and mammals it pushes up a part and then perforates it
centrally through an opening which, according to Keith, is
the pleuroperitonal opening, which may or may not close, just
as the processus vaginalis may or may not close.   As soon as
the lung is above the dorsal fascia, with its layer of transver-
salis muscle, it pushes its way forward into the transverse
septum in such a way as to separate this into a pleuropericar-
dial part and a pleuroperitoneal part, the latter being pushed
down to form the dorsal part of the definitive diaphragm.

   This idea is well supported by the facts of comparative an-
atomy.   Actual perforation of a tissue layer is, however, a
rather unusual embryologic principle, and it is in this respect
that the analogy of the descent of the testicle ceases, since this
organ is from first to last retroperitoneal and never perforates
a layer.   Also this theory does not help to solve any of the
problems of diaphragmatic hernia and assumes a contemporary
formation of pleuropericardial and pleuroperitoneal mem-
branes, which so far as their closure indicates, is not in ac-
cord with the facts in mammals.

CLASSIFICATION OF DIAPHRAGMATIC HERNIAS.

   The lack of any uniform method of classification of these
hernias has led to a great discrepancy and disagreement as
to the real meaning of the terms "congenital" and "acquired"
in the statistics reported by different authors.   Surgeons have
divided them surgically and judged the frequency of the va-
rious forms solely from their incidence at the operating table,
while anatomists have classified them anatomically on the basis
of autopsy findings.

   The following classification combines most satisfactorily the
essential features of this form of hernia:

   1. True hernias (those with hernial sack).
      A. Congenital (present at birth).
      B. Acquired.   a. Through the natural openings (mostly
         esophageal).   b. Elsewhere (traumatic or nontrau-
         matic).

2. False hernias (those without hernial sack).
   A. Congenital.
   B. Acquired (all traumatic).
3. Eventration of diaphragm.

This classification does not differentiate between those cases in which a congenital weakness may have been present at birth but in which the hernia did not manifest itself until later in life. Usually it is impossible to make such a differentiation, except in those cases in which no sack is present. If trauma can be excluded, such cases are all congenital. Where any prolonged intraabdominal pressure finally results in a hernia, there is no way of excluding a preexisting hernia.

This article is not concerned with hernias of traumatic origin. They are of interest primarily from the surgical point of view, are all acquired and bear no relation to the development of the diaphragm. They are not, however, all false, as some authors state, as a crush or fall may easily cause a tear in the diaphragmatic muscle without injuring either pleura or peritoneum, giving rise later to a true hernia whose traumatic origin has been forgotten. Such cases may heal and on subsequent discovery be indistinguishable from the congenital form, since the edges of the torn pleura and peritoneum readily grow together and form a smooth edge closely simulating a congenital opening. It is more usual for trauma to rupture both membranes as well as the muscle.

Diaphragmatic hernia has long been known to medical literature, the first congenital case being reported by Riverius in 1689. In 1824 Sir Astley Cooper gave a masterful account of a case. Since then cases have been reported more and more frequently, the condition has been suspected and sought for more often, and the diagnosis more frequently made antemortem. The question of why such hernias occur, why some types are more frequent than others and why some situations are rare and some common, it is the aim of this paper, so far as possible, to answer.

ETIOLOGY OF DIAPHRAGMATIC HERNIAS.

The factors involved in the occurrence of these hernias are as varied as the types themselves and differ according to the location of the hernia. There are, however, certain general

principles governing the occurrence of all forms, which may be divided into two groups, intrafetal and extrafetal.

1. Intrafetal.—These factors are concerned with the development of the fetus itself. The most important is the interference with the normal closure of the pleuroperitoneal membrane, resulting in a smaller or larger defect, usually posteriorly and without a sack. What prevents the closure of this membrane is a much mooted question. That it may be a simple retardation or cessation of growth (as in the case of a persistent foramen ovale) is shown by the frequency with which these hernias are associated with other congenital anomalies, such as a cleft palate, patent ductus arteriosus, other hernias, accessory lungs, and abnormal mesenteric relations of an atavistic nature. In other cases the defect is quite clearly a halt in normal development independent of other factors.

Another frequently mentioned cause is the premature prolapse of abdominal viscera into the thoracic cavity. The reasons given for this prolapse are: (1) abnormal decrease in intrathoracic pressure with a similar increase in intraabdominal pressure; (2) an abnormally long or free mesentery which allows the viscera access to the chest. These reasons do not bear close scrutiny, since it has been shown, first, that so long as the amniotic fluid is present there is no difference between the thoracic and the abdominal pressure, and second, that at the time the diaphragm is being formed the body walls are incapable of sustaining or exerting any pressure whatsoever. Jahn[3] has shown that certain normal changes in mutual position and relation of the large intestine and stomach have already taken place before the normal time of closure of the pleuroperitoneal membrane. Since these changes take place only in the abdomen the viscera could not have entered the chest until after the normal time of closure of the membrane, and hence failure of the latter to close was not due to any premature prolapse of the viscera. The fact that still further mesenteric changes normally taking place in the abdomen do not occur in the chest shows that the previous change occurred while the intestine was in the abdomen.

One other factor may increase the intraabdominal pressure and, by exerting pressure on the pleuroperitoneal membrane sufficient to shut off its normally poor blood supply (Vogel),[4]

cause a delay in its development. This is the occasional failure of the small bowel to enter the umbilical coelom, a condition which would considerably increase the abdominal contents at this time.

These factors concern the false type of congenital hernia. The true congenital hernias are due to an abnormal development or formation of the diaphragmatic muscle, and any process which retards or prevents this may easily lead to a herniation at the point of defect. Why the muscle fails at times to develop is not certain. It may be a question of local pathology, some interference with circulation or some fault in innervation. The defect is more usually a pathologic than an embryologic one.

Abnormalities in the growth of the liver have been frequently assigned as causes of a failure in the separation of the two cavities. Since a small liver has been mentioned as often as a large one and since both types have frequently been found accompanying the hernia, it is very doubtful if this organ plays any very important role in the development of the dorsal diaphragm.

2. Extrafetal.—These factors are the same as those associated with many other developmental defects, such as trauma to the mother and intrauterine inflammation. The inverted position of the fetus has been suggested as likely to favor the passage of the viscera from the chest into the abdomen, but such a position is not assumed till long after the normal closing period of the pleuroperitoneal membrane.

SITUATION OF THE HERNIA.

A consideration of any large number of reported cases of diaphragmatic hernia would lead one to believe that there is no portion of the diaphragm which is exempt and no natural opening which cannot, theoretically at least, serve as a point of weakness. As a matter of fact, these hernias fall into several distinct groups, some much more common than others, and there are certain points which never are the site of herniation. Thus the aortic opening has never been known to contain a hernia, first, because this opening is, anatomically and developmentally, not in the diaphragm but behind it;

second, its ring is tendinous, not muscular, and attached closely to the vertebræ by the crura on each side. The aorta is occasionally included in the hernial opening, but this is the result of the extreme defect of the diaphragm on one side and not of any herniation through the aortic opening itself. Similarly the quadrilateral foramen, through which passes the inferior vena cava, has never been found to be the site of a hernia. The intimate attachment of the vein walls to the central tendon and the broad expanse of liver below and around it render access of the abdominal viscera impossible.

The various hernial sites may be divided as follows: Anterior, central, posterior, esophageal, others.

Any of the first three types may be right or left, the latter being much the more frequent, as shown below.

Anterior.—These hernias are situated to one side of the diaphragmatic attachment to the sternum and occur almost entirely through Larry's space or the foramen of Morgagni, one of the socalled points of weakness, where there is a triangular muscular defect between the sternal and costal attachments of the muscle. These are known as parasternal hernias and are usually small defects, being the result of weakness at a point normally somewhat deficient in musculature. The possibility of an incomplete closure of the pericardial ventral recesses when the vitelline veins expand laterally can be excluded by the fact these hernias never communicate with the pericardial cavity. Though the muscle is deficient at birth, these hernias are practically all acquired as the result of a prolonged increase in intraabdominal pressure and are found almost exclusively in adults. The defects may be crossed like a griddle by muscle fibers which occur more commonly and numerously in younger individuals. The ratio of the size of these "spaces of Larry" to the size of the body is, as shown by Thoma,[5] practically the same in the newborn as in adults. Why then does this type of hernia not appear in the newborn in which hernias occur at far less favorable sites? There is proportionally less muscle in the adult spaces, the angle of the diaphragm with the sternum is less acute, the mesentery is longer and the liver is relatively smaller, but none of these factors seems to furnish a satisfactory answer. The almost universal presence of a sack renders the problem far different

from that concerned with the closure of the pleuroperitoneal canals.

These parasternal hernias are occasionally bilateral.   One has been reported by Lubosch[6] in which a double sack passed upward on either side of the falciform ligament and extended up into the anterior mediastinum.   This is an example of a congenital weakness which became an acquired hernia and is best explained by assuming that when the peritoneum folds in to separate the liver from the diaphragm (Fig. 8B) it meets some abnormal resistance from a thick falciform ligament which deviates the folds upwards toward the thorax to form two peritoneal pockets on each side.   Moser reports a case in which the hand could be passed into such a hernia on the left side through an easily reducible epigastric swelling and the entire apex of the heart grasped, undoubtedly through a greatly dilated left foramen Morgagni.

For such of these hernias as have no sack there seems to be only the explanation of a previous forgotten trauma.

CENTRAL· HERNIAS.

On first consideration these hernias do not readily lend themselves to any embryologic explanation.   They occur both with and without a sack at points unrelated to the fusion lines of the constituent elements of the diaphragm.   Those with a sack must be due to some local pathologic condition in the muscle; they are far less common than the false type.   These latter are most readily explained by assuming that the posterior part of the pulmonary ridge has begun to develop toward the longitudinal mass and has then for some unknown reason ceased to grow.   This would leave a ridge of tissue into which vertebral muscle fibers could advance.   As this ridge, together with the rest of the diaphragm, increases in size the defect between the two will move forward and take up a more central position relative to the rest of the diaphragm.   If there is no growth at all in the pulmonary ridge then the defect is a pure posterior one occupying the original pleuroperitoneal canal. The direction, the rate and the character of growth of the muscular diaphragm determine the eccentric or central position of these defects and whether they lie in the muscular or tendinous portion.   Thus most of the false hernias in the cen-

tral portions of the diaphragm were originally posterior de-
fects which have changed their relative position.

POSTERIOR.

These hernias show more variations in size than any of
the others. The majority of them are due to failure in the
closure of the pleuroperitoneal canals, hence they originate
in the lumbar region between the vertebral and costal portions
at the point where the pleuroperitoneal membrane last closes.
It has been repeatedly stated that this point is identical with
the socalled foramen Bochdalecki in the lumbar triangle, where
there is a normal deficiency of muscle much as in the para-
sternal spaces. This, however, is not the case. This "fora-
men" is simply a point of incomplete fusion of the vertebral
and costal muscle groups and does not appear until long after
the pleuroperitoneal canal has closed. That the two do not
even occupy the same spot is proved by the occurrence of a
posterior membranous defect with a normal "foramen" in a
single individual and on the same side, and a case has been
reported (Otto[7]) in which a left false and a right true pos-
terior hernia were present simultaneously.

According to Keith,[2] such posterior defects arise from a
failure of the point of perforation made by the lung to close
after this organ has made its way upward through the dorsal
fascia posterior to the transverse septum.

ESOPHAGEAL.

This situation is regarded by many surgeons as the most
common site for diaphragmatic hernia. In the living adult,
as seen at operation, it doubtless is, but in the sum total of
these hernias the esophageal ones are extremely uncommon.
Strictly speaking, there is no "esophageal opening"—that is,
the digestive tract is everywhere intimately enclosed by the
dorsal, and above the diaphragm, by the ventral, mesentery
through which the esophagus passes. The only "opening" is
that created by the failure of the diaphragmatic muscle to
closely surround the esophagus. The more the muscle fails
to do this the greater is the amount of the membranous par-
tition composed of peritoneum, pleura and interstitial tissue
which, under certain conditions, may become the site of a
hernia. For this reason esophageal hernias are commonly

of the true type, contained in a sack formed of relaxed membranous diaphragm intimately attached to the esophagus itself. Indeed, without the assumption of some forgotten trauma it is difficult to explain an esophageal hernia in which there is no sack present. The great majority of them, as found in adults, are of the acquired type and result from long continued coughing or straining in an individual whose esophageal muscular opening is abnormally large or weak. The congenital cases with a sack may be explained as suggested by Bund, by an abnormal development of the omental bursa (Fig. 9). If for any reason the infracardial bursa, which at an early stage communicates with the omental bursa and hence with the general abdominal cavity, should persist and remain open below, there would then be formed a direct passage beside the esophagus and through the diaphragm. This would afford an extremely vulnerable spot for a viscus to force its way upward along and close to the esophagus, through the muscular ring, simulating in every way a general dilatation of the opening. Since the stomach would be the organ most likely to herniate by this route, the esophagus would simply be carried upward at the fundus of the sack.

This infracardial bursa persists more frequently on the right than the left, so that we should at first expect that most esophageal hernias would be found in the right chest, but once the opening has become sufficiently dilated to allow the stomach to leave the abdominal cavity other factors control the side to which it goes. Thus the presence of the heart on the left would have a tendency to force the hernial sack to the right, as would also the greater negative pressure in the right thorax. An interesting case in point is that described by Rischbieth[8] in a newborn child with an encephalocele and other congenital abnormalities. There was a complete absence of the left half of the diaphragm save for a musclar strand to the left of the esophagus. There was also a huge dilated esophageal opening with a sack containing the stomach and almost all the other viscera which had herniated into the right chest and posterior mediastinum. The question arises why these viscera did not enter the much more accessible left chest which contained only the left kidney. Rischbieth explains the extensive herniation into the esophageal sack by the fact that

in the absence of a left diaphragm the descent of the right
diaphragm (which occurs as a result of simple muscular con-
traction, not as a part of respiration) caused a great increase
in the negative pressure of the right side and hence favored
the aspiration of the viscera toward that side.

The formation of the sack is somewhat obscure in those
cases where its neck is reflected not from the cardia but from
the pyloric portion of the stomach, the latter being to all
intents a thoracic or mediastinal organ and situated entirely
above the diaphragm. In such cases the stomach may never
have been in the abdomen, but, developing at an abnormally
high point in the digestive tract, may have remained station-
ary while the diaphragm was formed and closed in its normal
position. There is no reason why the pleuroperitoneal mem-
brane might not close posteriorly, even with the stomach in
such a position, since its relation to the dorsal mesentery would
be entirely normal. Where there is a large sack reflected from
the cardiac end of the stomach, the cause is clearly one of
herniation from below and is certainly the more common type.
The difficulty of reducing many of these hernias suggests a
high mesenteric attachment of the stomach when adhesions
between it and the thorax can be excluded.

OTHER SITUATIONS.

A few rarer points of exit are by way of the sympathetic
nerve chains in the crura of the disphragm and via the azygos
minor vein. Another rare but extremely interesting point of
herniation is the floor of the pericardium. Keith[9] reported
two cases of this type in 1910, Martland[10] one in 1908. All
three were in adults who had lived for years with the heart
surrounded by coils of small bowel without any apparent
symptoms. Such hernias, which are of the false type without
a sack, result, according to some authors, from a congenital
failure in closure of the pleuropericardial membrane. Since,
however, there was no communication between the pleural and
pericardial cavities, this seems a hardly tenable theory. A
more plausible one would explain the defect on the basis of a
failure in complete development of the transverse septum, this
being the structure which forms the adult separation between
the peritoneal and pericardial cavities. Were this earliest en-

largement of the adult diaphragm defective, one would certain-
ly expect a secondary defect in the subsequently formed pleuro-
peritoneal and pleuropericardial membranes, structures which
were entirely normal in the three cases. As no congenital
cases have been reported, more satisfactory explanation is
afforded by assuming all these adult cases to be the result of
some past trauma which ruptured the central tendon below
the heart. This is the explanation assumed by Stoeber in the
case of a similar hernia in a dog, because the edges of the
opening, instead of being firm, showed a free flaccid margin
of pericardioperitoneum which, when brought together, com-
pletely closed the opening, an unlikely condition in a defect
of congenital origin. Frank defects between the pericardial
and pleural cavities have been reported several times, but in
none of these was there any communication between the
pericardial and peritoneal cavities.

The above classification of the points of herniation does not
take into consideration one other frequent condition, namely,
a total unilateral lack of the diaphragm. Such cases if exam-
ined carefully will almost always show a small sickle shaped
fold of muscle anteriorly representing the transverse septum,
the defect being due to a complete failure of development of
the pleuroperitoneal membrane, which becomes proportionally
greater as the growth of the fetus continues. Total lack of
the whole diaphragm, which has been reported once, occurred
in a monstrosity. That left hernias are far more common
than right sided ones is a fact, the explanation for which has
long been a subject of controversy, though it is commonly
assumed to be the result of the greater protection offered by
the liver to the right side of the diaphragm. This undoubtedly
plays an important role in the matter, but it has been shown
that at the time of the development of the diaphragm the left
half of the liver affords the diaphragm about as much pro-
tection as the right. The foramen of Bochdalecki is larger
on the left than the right, indicating that muscle formation
is slower on the left side, but this does not explain the false
type of defect. The mobility of the stomach has been con-
sidered instrumental in exposing the left side, while the fur-
ther descent of the right lung into the abdominal cavity may
prevent the entrance of viscera. It has been shown above,

however, that a mechanical interference with closure on the part of the abdominal viscera plays but a small part in the etiology of early defects, so that there must be some other factor which at the very outset renders the left diaphragm more liable to incomplete development. Its exact nature must remain one of the unsettled problems of embryology.

CONTENTS OF THE HERNIA.

Lacher states that the only viscera which have not been found at least once in the chest cavity are the urogenital organs and the rectum. The stomach is, of course, the most frequent organ to herniate, the large intestine the next, and the small bowel next. In one instance only has a thoracic organ been found in the abdomen, in which case the tip of the right lung was found between the folds of the falciform ligament. The defect presumably involved the right foramen of Morgagni.

VISCERAL RELATIONS IN THE THORAX.

The extrusion of the abdominal viscera into the thorax has a marked effect on those which normally occupy the chest cavity. The heart is almost always displaced to the side opposite the diaphragmatic defect, and the lung on the same side is pushed upward and hindered in its development, especially where the hernia has existed from birth. It is often a difficult matter to decide whether the appearance of the lung is due to a pressure atrophy or whether there exists a primary failure in lung development of a truly embryologic character. A frequent finding secondary to the presence of the abdominal viscera in the chest cavity is a further herniation through the posterior mediastinal tissues of the organs occupying the apex of the sack. This may be so extreme as to permit viscera which herniated through the left diaphragm to lie entirely in the right chest. The same situation occurs less often in hernias through the parasternal spaces, where a sack originating on the left side may pass across the tissues of the anterior mediastinum and come to lie in the right chest.

It has been frequently observed that in the absence of a hernial sack the prolapsed viscera never becomes adherent to the parietal pleura of the chest walls. Since the transverse

colon, for instance, often lies in the chest at a time previous to that at which, if in the abdomen, it would become anchored at certain points to the parietal peritoneum, there must be some explanation as to why this fusion fails to occur in the thorax. The pleural membrane may be less suited to such a union; constant motion of the heart may be a factor.

PROGNOSIS.

A surprisingly large number of cases of diaphragmatic hernias in adults have been discovered accidentally at autopsy and were entirely unsuspected during life. So long as strangulation does not occur the presence of the abdominal viscera within the chest is by no means incompatible with life. Ulceration of the stomach in its new situation is not uncommon, Koenboek[11] in 1914 reporting 32 such cases. These ulcers have occasionally ruptured and given rise to pyothorax, not, as would seem at first more likely, to peritonitis, the abdominal cavity being protected by the contraction of the diaphragmatic muscular ring about the opening. Most of the newborn cases with posterior defects and no sack die shortly after birth; those with a sack are more apt to live than those without, as shown by the small number of adults who show a posterior or central defect without a sack.

These hernias are coming to operation more and more frequently. In 1912 Scudder[12] could find only 53 cases which had been operated upon; since then there have been a number of cases successfully cured by surgery, those in which the herniation takes place through a dilated esophageal opening giving the most satisfactory results. The true hernias are easier to close than the false ones. Many long standing cases are of course incapable of reduction, either by the thoracic or abdominal approach, but much progress has been made since the days when metallic mercury was given by mouth for the purpose of reducing a herniated stomach by gravity.

EVENTRATION OF THE DIAPHRAGM.

This condition is not, strictly speaking, a true form of hernia of the diaphragm, but it has so long been considered as such in classifications of these hernias that it is justifiable to include it in the present discussion, the more so as its etiology

is, in part at least, congenital. It consists essentially in a great thinning out and weakening of the musculature of the diaphragm on one side, almost always the left, so that the dome rises high in the chest, often up to the level of the second rib. The lung above is compressed; the abdominal viscera rise to occupy the space below. It can readily be seen that a differential diagnosis between this condition and a true hernia with a large sack is often very difficult and one which has provoked an extensive discussion in the literature.

CLASSIFICATION.

Eventrations are best classified etiologically as suggested by Lerche:

1. Congenital.
2. Acquired (a) acute, (b) chronic.

The chronic cases are the uncured acute ones, the latter usually resulting from some pathologic condition, such as an acute infectious disease, affecting either the diaphragmatic muscle or the phrenic nerve. The question of etiology has given rise to a considerable difference of opinion and numerous arguments have been advanced in support of various theories.

Many authors claim that the majority of the cases are congenital, with the diaphragamtic muscle on one side atrophied at birth. This view is based: (1) on the occurrence of the condition in the newborn, in conjunction with other fetal abnormalities; (2) on the fact that most of the cases are left sided and in this respect resemble congenital hernia of the disphragm; (3) on the absence of signs of pressure on the lungs, showing that the latter never attained full development, being small but normal in formation. On the other hand, opponents of this view cite the cases which have followed some definitely pathologic condition, either of a general nature, like typhoid fever, or local, as in empyema. Increased intra-abdominal pressure, spinal lesions involving the phrenic nerve and idiopathic muscular atrophy have also been assigned as causes. In all these it is of course difficult to disprove the contention that the eventration was present but unsuspected previous to the onset of the subsequent illness.

The bulk of the evidence seems to favor a congenital origin. In all lesions of the phrenic nerve, aside from local pathology as in one case of a suppurating lymph node, there is no reason why the left side should be so much more often affected than the right.   The same holds true of general infectious diseases and muscular atrophies.   The fact that the symmetry of the chest wall is not disturbed, except in cases of local thoracic suppuration, is another point in evidence of the fact that the eventration has been present from birth, since a later change in muscle tension on one side would show itself in an asymmetry of the chest wall.

If simple prolonged increase in intra-abdominal pressure were an important factor, then we should expect to find eventration more commonly in women than in men, whereas statistics show exactly the reverse.   Failure of the lung to develop to its normal size and so to exert normal pressure against the diaphragm may allow a premature stretching of the developing muscle, though the latter condition may be the cause of failure in lung development.   On the other hand, the almost universal finding of a dextrocardia suggests the mechanical effect of an acquired lesion, since congenital dextrocardia is rare and would be unlikely to accompany so large a proportion of diaphragmatic eventrations.   They may both be congenital manifestations of a retarded development.

Microscopic examination of the disphragm in these cases reveals an atrophy of the muscle fibers and also an aplasia of them.   The phrenic nerve may also show a pathologic change, either secondary or primary, so that there may be an atrophy of the nerve with secondary aplasia of the muscle or vice versa or both.

It has been suggested by Weigert that an injury may occur to the phrenic nerve during birth, as in a brachial paralysis, but this fails to explain the frequency with which the left side is involved.

In 1915 Bayne-Jones[13] collected 45 cases of eventration, most of which had been proved by autopsy.   Of these, 8 were females, 37 males.   The ages ranged from birth up to 75 years, but the majority were in adults.   There were 7 cases associated with other congenital anomalies and 9 were classified as acquired, the rest as congenital.

We have collected below the cases reported since 1915. As
a number of these have been proved by X-ray only, they are
somewhat less authentic than those confirmed by autopsy or
operation. This latter procedure has been attempted once with
reported success, but eventration is generally considered to be
a nonsurgical condition.   It is certainly not comparable in this
respect with diaphragmatic hernia.   One case of spontaneous
cure has been reported and confirmed by serial X-ray exam-
inations.

It had been considered possible that this investigation of
the diaphragm might throw some light on the question of
cardiospasm and esophageal stricture.   Beyond the suggestion
that the former may be due to a spasm, not of the esophageal
muscle but to that of an abnormally tonic or spastic diaphragm
and the latter to the scar tissue following an unrecognized and
healed rupture of the esophageal "opening," no further con-
nection between these subjects has been observed.

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A complete bibliography comprising about 300 references will be
furnished on request.

TABULATION OF NONTRAUMATIC DIAPHRAGMATIC HERNIAS SINCE 1900 (Fig. 11)

No.	Year	Author	Sex	Age	Side	Type	Size (cm)	Position	Congenital or Acquired	Remarks
1	1904	Planchu	M	Newborn	Left	False	2cm diam.	Central	Congenital	
2	1904	Planchu	M	Newborn	Right	False	2cm diam.	Posterior	Congenital	
3	1921	Hirsch	M	36	Left	True	?	?	Acquired	Diagnosis proved by X-ray. Stomach not in chest.
4	1905	Herz	M	30	Left	?	?	?	Congenital	Ruptured stomach.
5	1906	Daxenberger	M	23	Left	False	2 small ones	One central near esophagus	Congenital plus pathologic rupture	Stomach in right posterior mediastinum.
6	1918	Bund	?	8 months	?	True	?	Dilated esophageal opening	Congenital	
7	1913	Rischbieth	?	Newborn	Left	False / True	?	Total left defect. Dilated esopha-	Congenital	Hernia passed up through ligamentum pulmonale.
8	1913	Rischbieth	F	82	?	True	?	Esophageal opening dilated.	Acquired	Cured by operation.
9	1903	Liepman	M	Newborn	Left	False	10x3	Posterior	Congenital	
10	1920	Rowlands	M	19	Left	True	Small	Posterior	Acquired	Sack in folds of suspensory ligament of the liver.
11	1915	Grules	?	Newborn	Left	False		Central	Congenital	
12	1914	Skillern	M	55	Right	True	Small	Parasternal	Acquired	Diagnosed by X-ray only.
13	1903	Lepage	?	Newborn	Right	False	Large	Posterior	Congenital	Acute strangulation with death.
14	1916	Freud	M	38	Right	True	?	Para-esophageal	Acquired	Diagnosed by X-ray only.
15	1910	Marie	F	28	Left	True	?	Posterior	Congenital	Acute strangulation.
16	1910	Dietler	F	16	Right	?	5cm diam.	Posterior	Congenital	Diagnosed by X-ray only.
17	1916	Vitrac	M	?	Left	False	?		Congenital	Diagnosed by X-ray only.
18	1917	Weinberger	M	31	Right	False	?		?	Diagnosed by X-ray only.
19	1915	Monti	?	4 mos.	Left	?	?		Congenital	
20	1921	Allard	M	?	Left	?	3cm diam.	Central	Congenital	Foramen Bochdalecki distinct from defect.
21	1916	Ruder	?	Newborn	Right	False	10cm diam.		Congenital	
22	1914	Gruber	M	48	Right	True	3cm diam.	Posterior	Acquired	Further herniation of stomach into right flank to stimulate pyonephrosis. Many ulcers and adhesions.
23	1905	Hamdi	M	50	Right	True	3cm diam.	Posterior	Acquired	
24	1921	Jahn	F	Fetus	Left	False	3x5cm	?	Congenital	Scoliosis. Abnormal secondary herniation from left to right chest through mediastinum.
25	1914	Cailloud	M	48	Right	?	Large	Central	Of Trauma ?	Liver and colon in right chest.
26	1911	Scholz	?	Newborn	Right	True	4cm diam.	Para-esophageal	Congenital	
27	1920	Scudder	M	16	Right	True	?	Dilated esophageal opening	?	
28	1920	Huffman	F	Adult	?	True	?		Congenital	Other hernias, abnormalities of blood vessels, only 11 ribs, patient fora- men ovale. No symptoms.

TABULATION OF NON-TRAUMATIC DIAPHRAGMATIC HERNIAS SINCE 1900 (Fig. 11)—Continued.

No.	Year	Author	Sex	Age	Side	Type	Size (cm)	Position	Congenital or Acquired	Remarks
29	1920	Mathews	F	Adult	?	True	?	Dilated esophageal opening	Congenital weak. Acq. hernia	Sack in right posterior mediastinum. Symptoms 10 years.
30	1919	DeCourcy	F	25	?	True	?	Dilated esophageal opening	?	Marked anemia. Operation and cure.
31	1919	Sorest	F	?	Left	?	?	Para-esophageal	?	Operation.
32	1912	Scudder	M	29	Left	False	?	Central	Congenital	Symptoms only recently.
33-38	1903	Hertz	?	Newborn	Left	?	?	No further data given	Congenital	Liver in chest.
39	1903	Hertz	F	Fetus	Right	False	Huge	Total half	Congenital	Patent foramen ovale.
40	1913	Potczkl	?	Newborn	Right	False	Large	Posterior	Congenital	Big liver. Common mesentery for small and large bowel.
41	1914	Gruber	M	9 days	Right	False	Moderate	Posterior	Congenital	
42	1914	Gruber	?	Newborn	Right	False	Large	Posterior	Congenital	Secondary hernia through posterior mediastinum.
43	1914	Gruber	F	Newborn	Left	False	Moderate	Posterior	Congenital	Myelocele. Club foot. Entire mesentery free.
44	1914	Gruber	F	Fetus	Left	False	2cm diam.	Posterior	Congenital	Free bowel mesentery.
45	1914	Gruber	M	Fetus	Left	False	3cm diam.	Posterior	Congenital	Free mesentery.
46	1914	Gruber	F	Newborn	Left	False	Large	Posterior	Congenital	Free mesentery.
47	1914	Gruber	M	Newborn	Left	False	Large 4cm diam.	Posterior	Congenital	Accessory left lung. Free mesentery.
48	1914	Gruber	F	7 days	Left	False	2cm diam.	Posterior	Congenital	Free mesentery.
49	1914	Gruber	F	Fetus	Left	False	2cm diam.	Almost whole half	Congenital	Degenerate adrenal. Encephalomyelocele. Free colonic mesentery.
50	1914	Gruber	M	Newborn	Left	False	Large	Whole half	Congenital	Persistent ductus arteriosus.
51	1921	Cohn	F	Newborn	Right	False	2cm diam.	Posterior	Congenital	Very large liver. Right lung embryonic.
52	1920	Mitchell	F	50	Left	True	15x20cm	Parasternal	?	Sack passed across midline to right chest.
53	1921	Bakes	M	43	Left	?	3x6cm	Posterior	?	Operation with freezing of phrenic nerve.
54	1905	Jonas	M	Newborn	Right	False	2cm diam.	?	Congenital	Liver very small.
55	1905	Jonas	F	Newborn	Left	True	2x3cm	Central	Congenital	
			F	Newborn	Left	False	1cm diam.	?	Congenital	
56	1908	Don	M	61	Left	True	?	Dilated esophageal	Congenital	Death due to intestinal obstruction.
57	1912	Morestin	M	38	?	False	?	Central	?	Operation followed by death. Type probably due to old trauma.
58	1903	De Cardinal	M	58	?	False	?	Pericardial	Acquired	None.

No.	Year	Author	Sex	Age	Side	True/False	Size	Location	Type	Notes	Remarks
59	1907	Risel	F	Newborn	Left	False	4x4½cm	Posterior	Congenital		Portions of liver tissue in sack wall, showing liver to be a factor in its development.
60	1907	Risel	F	Newborn	Left	True	Large	Whole half	Congenital		Whole stomach in thorax with pylorus coming through esophageal opening. Stomach in posterior mediastinum.
61	1905	Chartier	?	4 mos.	?	True	Large	Dilated esophageal Posterior	Congenital	Anomalies of vessels.	
62	1909	Bertier	F	Newborn	Left	False	?	Posterior	Congenital	Right lung underdeveloped.	
63	1909	Bertier	F	Newborn	Right	True	?	Whole half	Congenital		
64	1906	Cantley	?	4 mos.	Right	True	?	?	Congenital	Easily reducible epigastric swelling.	Liver and gall bladder in right chest.
65	1902	Moser	M	Adult	Left	True	?	Parasternal	?		Heart grasped by hand. No symptoms.
66	1904	Hunter	M	2 yrs.	Right	True	2x3cm	Posterior	Congenital		Sack contained stomach, not liver.
67	1907	Hunter	?	Newborn	Right	False	?	?	Congenital		
68	1907	Hunter	?	Newborn	Left	False	?	Posterior	Congenital		Secondary herniation through posterior mediastinum.
69	1908	Williams	F	9 mos.	Left		?		Congenital		
70	1915	Kakels	M	54	Left	True	Large	Posterior	Congenital	Abnormal liver. Dextrocardia. Stenosis of aorta.	No further data.
71	1921	Roos	M	35	Left	?	?	?	?	Patent foramen ovale and ductus arteriosus.	
72	1918	Gross	?	Newborn	Left	True	No left muscle	Whole half	Congenital		Herniation of spleen through posterior mediastinum into right chest.
73	1918	Fisk	M	Newborn	Left	False	Large	Posterior	Congenital	Lungs normal.	Strand of diaphragm across top of sack.
74	1913	Meyer	F	53	Left	True	7x6cm	Dilated esophageal	?		Transverse colon in pericardial sack; rest of diaphragm normal. No symptoms.
75	1908	Martland	M	70	Left	False	4x5cm	Pericardial	?		
76	1910	Bokay	?	10 days	Left	True	4cm diam.	?	Congenital	Left lung size of raisin, kidneys embryonic.	
77	1910	Bokay	?	11 mos.		True	?	Central	Congenital		
78	1911	Rawes	?	?	Left	False	3cm diam.	?	?		
79	1914	Monks	M	43	Left	False	8cm diam.	?	Congenital		No symptoms.
80	1918	Dickie	?	Adult	?	True	?	Dilated esophageal	Acquired	Complicated by intestinal obstruction.	
81	1920	Carmichael	?	Newborn	Left	?	?	Posterior	Congenital		
82	1921	Barnett	?	8 mos.	Left	True	Large	?	Congenital	Secondary mediastinal sack in which large bowel was strangulated.	
83	1916	Gordon	M	56	Right	True	Large	Anterior	Acquired	Death from acute obstruction.	
84	1913	Vogel	M	47	Left	False	15cm diam.	Posterior	Congenital		

TABULATION OF NON-TRAUMATIC DIAPHRAGMATIC HERNIAS SINCE 1900 (Fig. 11)—Continued.

No.	Year	Author	Sex	Age	Side	Type	Size (cm)	Position	Congenital or Acquired	Remarks
85	1907	Ulrich Walton	F	26	?	True	?	Dilated esophageal opening	?	Hernial sack in right chest.
86	1917	McCleave	M	3½ mos.	Left	False	2cm diam.	Posterior	Congenital	Death.
87	1916	Miller	F	50	Left	False	?	Central	Congenital	Death due to obstruction.
88	1919	De Buys	?	4 mos.	Left	False	?	Posterior	Congenital	
89	1914	Fletcher	?	2 yrs.	Left	?	2cm diam.	Central	Congenital	Death due to obstruction.
90	1917	Bevan	F	41	?	True	?	Dilated esophageal opening	?	
91	1917	Bevan	F	45	?	True	?	Dilated esophageal opening	?	
92	1920	Mayes	?	Newborn	Left	False	2x5cm	Posterior	Congenital	Death from asphyxiation. Patent foramen ovale and ductus arteriosus. Rudimentary left lung. Left kidney in chest.
93	1921	Hajek	M	46	Left	True	5cm diam.	Posterior	Acquired	Entire left diaphragm muscle poor. Died of strangulation.
94	1919	Pohlman	M	63	Right	False	8cm diam.	Central	Congenital	Marked cirrhosis of liver.
95	1921	FitzMaurice Kelley	M	35	Both	True	?	Parasternal	Acquired	
96	1918	Shafer	?	2 mos.	Right	False	3cm diam.	Central	Congenital	Left accessory lungs (two). Accessory spleen.
97	1917	Cockayne	?	Fetus	Left	False	2.5cm diam.	Posterior	Congenital	Sack in anterior mediastinum.
98	1918	Lubosch	M	Adult	Both	True	1cm diam.	Parasternal	Acquired	
99	1914	Rise	M	71	Left	False	2½x2cm	Posterior Anterior	Congenital	
100	1918	Gross	M	53	Left	False	?	parasternal ?	?	Strangulation.
101	1903	Chadburn	M	30	Left	False	8x12cm	Central	Congenital	Death due to sudden gastric distension.
102	1907	Williams	M	77	Left	False	4cm diam.	Posterior	Congenital	No symptoms.
103	1911	Harris	F	31	Left	False	Whole half	?	Congenital	Died of obstruction following birth of child.
104	1910	Ringrose	F	28	Left	False	8cm diam.	Posterior	Congenital	Whooping cough a possible added factor.
105	1922	Borden	F	25	Left	False	7cm diam.	Central	Congenital	
106	1918	Downes	M	7	?	False	3cm	Dilated esophageal opening	?	Reported as congenital but had a history of trauma.
107	1909	Bechman	?	17	Left	False	?	Posterior	Congenital	Symptoms for 18 months only.
108	1909	Bechman	?	47	Left	False	?	Dilated esophageal opening	Congenital	
109	1909	Bechman	?	47	?	True	?	Dilated esophageal opening	Acquired	
110	1920	Keith	?	17	Right	True	?	Posterior	Congenital	
111	1917	Rautenberg	?	?	Left	?	?	Posterior	?	

No.	Year	Author	Sex	Age	Side	True/False	Size	Location	Type	Remarks
'12	1916	Seibert	F	68	?	True	?	Dilated esophageal opening	Acquired	History of three difficult labors. Viscera were in left thorax.
113	1915	Kilner	M	30	Right	?	?	Parasternal	Acquired	Gas between fold of falciform ligament which also contained the tip of right lung. Only case of thoracic viscera entering abdomen.
114	1919	Warren	?	16	?	True	?	Dilated esophageal opening	?	Hernia followed a left empyema two years before. Hernia diagnosed as ulcer and gastroenterostomy performed. Hernia shown by X-ray three months later.
115	1919	Warren	?	30	?	True	?	Dilated esophageal opening	?	
116	1917	Kohler	F	12	Left	True	?		Acquired	
117	1904	Taylor	F	Newborn	Left	True	Entire half	?	Congenital	
118	1904	Gifford	F	4 mos.	Right	False	3cm diam.	Posterior	Congenital	
119	1904	Gifford	F	4 mos.	Right	False		Central	Congenital	
120	1921	Cleiz	F	Newborn	Left	False		Posterior	Congenital	
121	1913	Reiss	?	3 yrs.	Left	True	Large	Central	Congenital	Huge liver. Duodenum with a free mesentery.
122	1920	Grelve	F	5½ yrs.	?	True	5x6cm	Dilated esophageal opening	?	History of difficult labor and prolonged cough.
123	1912	Waille	M	24	Both	True	?	Parasternal	Acquired	Cough for 5 years. Repaired by operation. Both hernias repaired by operation.
124	1920	Foster	M	24	Left	False	?	?	Congenital	
125	1911	Mercade	F	27	Left	?	3cm diam.	Posterior	?	Traumatic perforation of stomach in chest. Perforated stomach ulcer with pyothorax.
126	1908	Courmont	M	32	Left	?	5cm diam.	Central	Acquired	Hernia followed a left empyema.
127	1904	Davergne	?	7	Right	False	3cm diam.	Posterior	Congenital	
128	1912	Devraique	?	Newborn	Left	False	Huge	Posterior	Congenital	Other abnormalities including undescended testicle.
130	1911	Keck	M	Newborn	Left	True	3x4cm	Anterior epigastrium	Congenital	Epigastric tumor with absence of lower sternum and anterior diaphragmatic muscle.
131	1921	Bergman	M	Adult	?	True	?	Anterior parasternal ?	Congenital & Acquired	Recent strangulation.
132	1908	Patel	M	69	Left	False	5x4cm	Anterior parasternal ?	?	Secondary protrusion of intercostal wall producing a visible tumor. Cardiac history with prolonged cough. Also an umbilical hernia.
133	1909	Gret	M	58	Right	False	7x6cm		?	Abnormally large liver.
134	1908	Berthaux	?	Newborn	Left	False	Huge	Posterior	Congenital	Numerous other hernias in family, including father and mother.
135	1908	Schreiber	F	Newborn	Left	False	Huge	Posterior	Congenital	
136	1918	Du Pan	F	63	Left	True	3cm diam.	Central	Acquired	Previous repair of ventral hernia, a possible factor in increasing abdominal pressure.
137	1918	Montandos	M	41	?	True	?	Dilated esophageal opening	Acquired	Severe strain a factor. Operation and death.

TABULATION OF CASES OF EVENTRATION SINCE 1915.

No.	Date	Author	Sex	Age	Side	Symptoms	Pathology Remarks
1	1920	Funk	F	39	L	Pain and vomiting.	Diagnosed by X-Ray.
2	1917	Neuman	M	44	L	History of pneumonia.	Atrophy of muscle and phrenic nerve.
3	1918	Aronson	F	20	R	History of asthma, pain in chest, vomiting.	Congenital megacolon; phrenic paralysis from trauma. Operation for megalocolon.
4	1921	Korns	M	20	R	No history.	Aplasia of right lung, dextrocardia, hypospadias and undescended testicles.
5	1918	Assman	M	38	L	Sense of pressure in chest.	
6	1922	Langlen	F	10	L	Loss of weight.	Cured by operation with excision of centre of left diaphragm.
7	1920	Weigert		2 mo.	L	Case of high forceps.	Cured in four months. Checked by X-Ray. ? of birth paralysis of phrenic nerve.
8	1919	Schwenke	M	23	L	Palpitation, pain in left side.	Diagnosed by X-Ray.
9	1919	Schwenke	M	30	L	Dysphagia.	Operation for hour glass stomach.
11	1917	Betchov	M	46	L	Cough, dyspnea.	Diagnosis by X-Ray.
12	1916	Wood	M	18			
13	1917	Minkowski	M	47	L	Vague symptoms for 2 years.	Severe trauma at seven years of age.
14-20	1916	Well				Five indefinite cases of left eventration without sufficient data to tabulate, and diagnosed by X-Ray only.	
21	1922	Lercher		37	L	Chronic cough.	Due to suppuration in lymph node involving phrenic nerve. Operation and cure.

Total since 1915............. 21

Right 2
Left 17
Indef. 2

TABULATION OF CASES REPORTED BY THOMA AND GROSSER
UP TO 1900.

1. False Diaphragmatic Hernia (nontraumatic):
   a. Left—
      1. Total lack.............................27 (all congenital)
      2. Central defect ....56 (29 adults, 5 children, 22
                         newborn)
      3. Posterior defect.................17 (3 adults, 13 newborn, 1
                         child)
   b. Right—
      1. Total lack.............................. 5 (4 newborn, 1 child)
      2. Central defect..................18 (8 adults, 2 children, 8
                         newborn)
2. True Diaphragmatic Hernia (nontraumatic):
   a. Left—
      1. Anterior (parasternal...... 6 (all adult)
      2. Central .... 7 (6 adults, 1 newborn)
      3. Posterior .... 1 (1 child)
   b. Right—
      1. Anterior (parasternal).... 6 (all adult)
      2. Central .... 2 (both newborn)
      3. Posterior .... 1 (adult)
   c. Esophageal .... 4 (1 child, 3 newborn)
3. Eventration:
      1. Left 7
      2. Right 1
4. Location Indefinite:
      1. Left 61
      2. Side not mentioned..........27
5. Totals:
      1. Left 175   Adults 61
      2. Right 42   Children 13
      3. Eventration 8   Newborn 89
      4. Esophageal 8
                    True 27
                    False 137

STATISTICS ASSEMBLED FROM CASES SINCE 1900

Age	Newborn	Children	Adults	
Total	50	10	58	
Percentage	42.4%	8.6%	49%	
Sex	Male	Female		
Total	53	35		
Percentage	60%	40%		
Sack Present				
Total	Newborn	Children	Adults	Indefinite
49	9	3	31	6
Percentage				
42.2%	18.3%	6.1%	63.5%	12.1%
Sack Absent				
Total	Newborn	Children	Adults	Indefinite
67	38	3	22	4
Percentage				
57.8%	56.7%	4.5%	32.8%	6.0%
Adults				
	Sack	No Sack		
Total	31	22		
Percentage	58.5%	41.5%		
Children				
	Sack	No Sack		
Total	3	3		
Percentage	50%	50%		
Newborn				
Total	9	38		
Percentage	19.1%	80.9%		

Location of Defect:

	Left	Right	Anterior	Posterior	Central	Esophageal
Total	82	33	12	42	21	20
Perc'ge	71.3%	28.7%	12.4%	45.5%	22.1%	21%

(Exclusive of three cases of total lack of one-half the diaphragm.)

These statistics are necessarily somewhat inaccurate, since each item of data was not always given in each case, but they serve to show the relative frequency with which these hernias occur, both as regards their situation and their type.

FIGURE 1.

Diagram illustrating formation of the heart in the guinea pig. The splanchnic mesoderm over the vitelline veins is migrating medially to fuse with the same layer on the other side.

(After Strahl and Carius.)
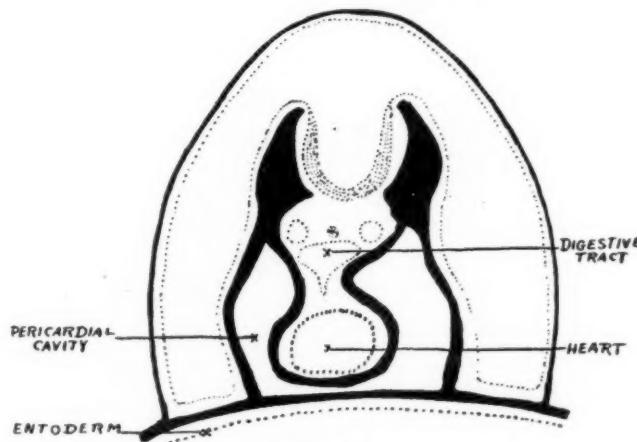
FIGURE 2.

Later stage of Fig. 1. The two vitelline veins have fused to form the heart and the fusion of the splanchnic mesoderm of each side has formed the pericardial cavity. (After Strahl and Carious.)
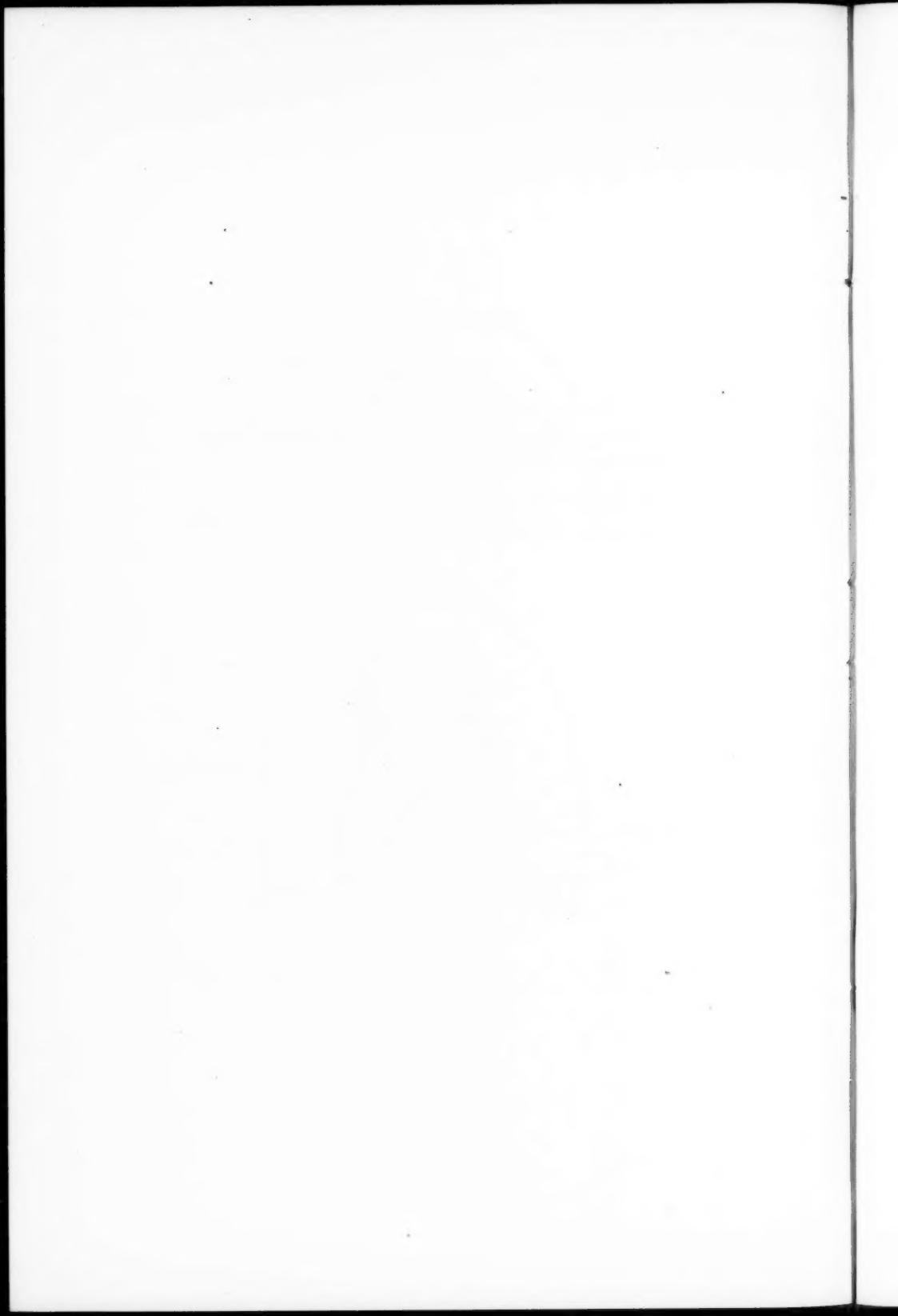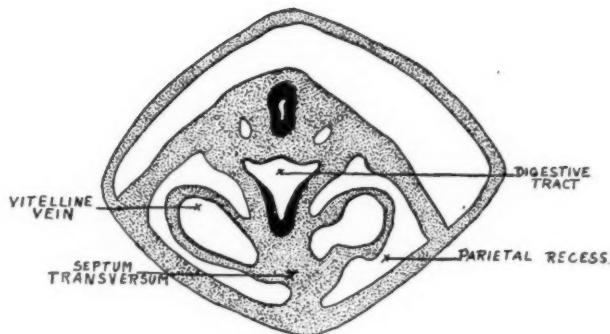
FIGURE 3.

Diagram to illustrate the formation of the lateral portions of the septum transversum by the outgrowth of the vitelline veins toward the body wall and into the parietal recesses. (Cross section of rabbit after Ravn.)
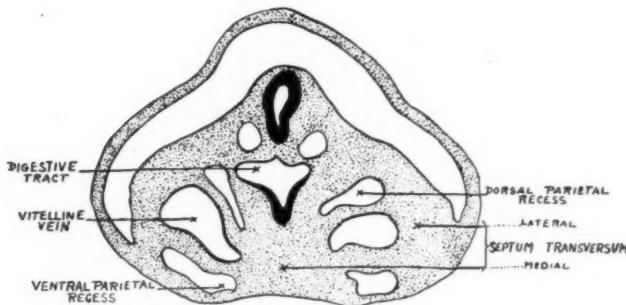
FIGURE 4.

Later stage of Fig. 1. The vitelline veins have fused with the body wall, forming the lateral portions of the septum transversum and dividing the parietal recesses into dorsal and ventral recesses. (Ravn.)

A B
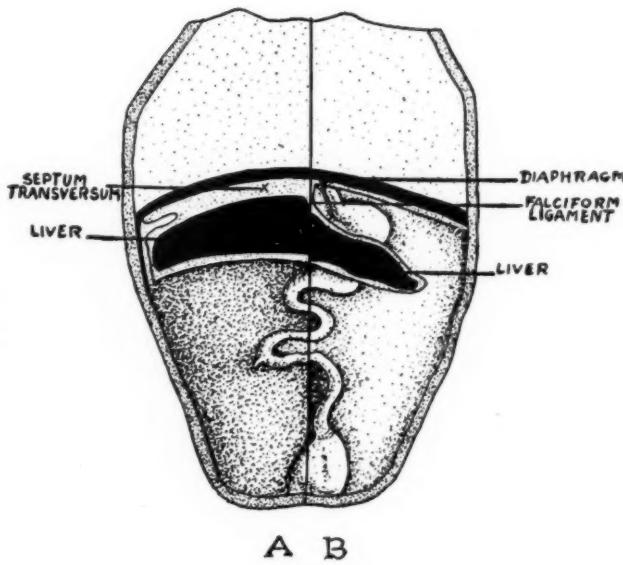
FIGURE 5.

Diagram showing the liver enclosed within the transverse septum and the manner in which infolding of the peritoneum constricts off the liver and forms the diaphragm and falciform ligament.
A.—Early stage. B.—Later stage. (Modified after McMurrich.)
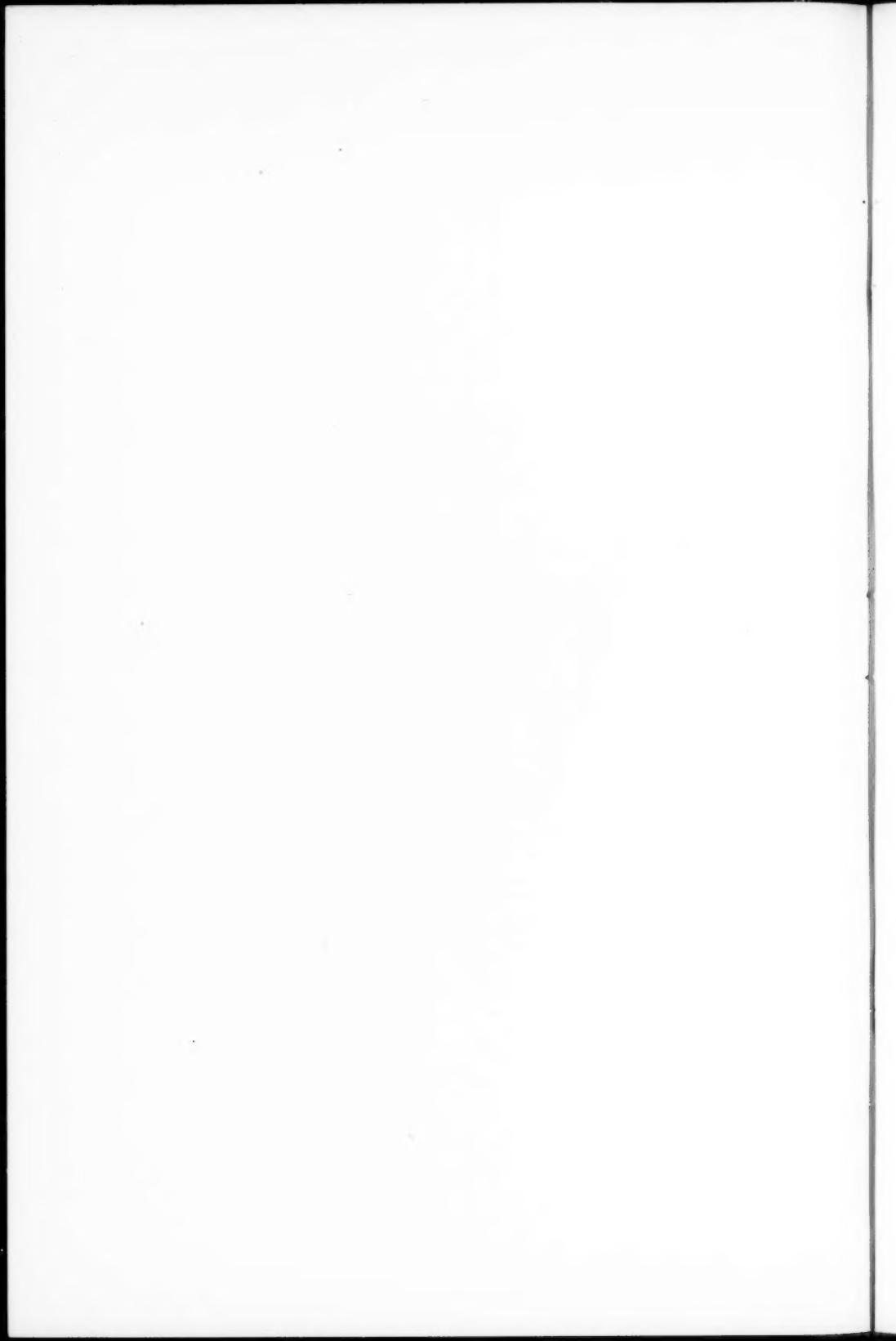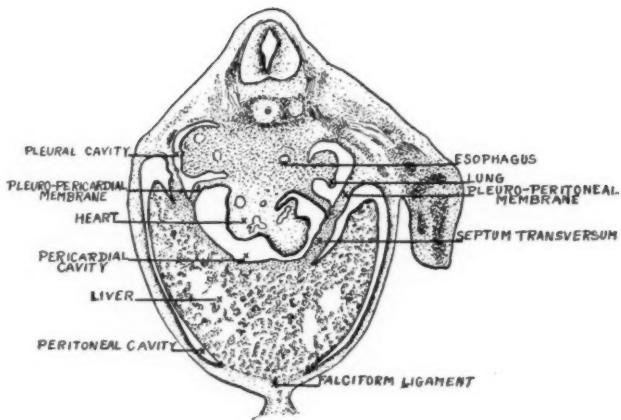
FIGURE 6.

Transverse section of a 10 mm. human embryo showing the pleuropericardial and pleuroperitoneal membranes separating the pericardial, pleural and peritoneal cavities. (After Prentiss.)

FIGURE

Transverse section in a ... human embryo showing the ... and the mesopneumonia? membranes separating the ... pleural, dorsal and peritoneal cavities. (After Bremer.)
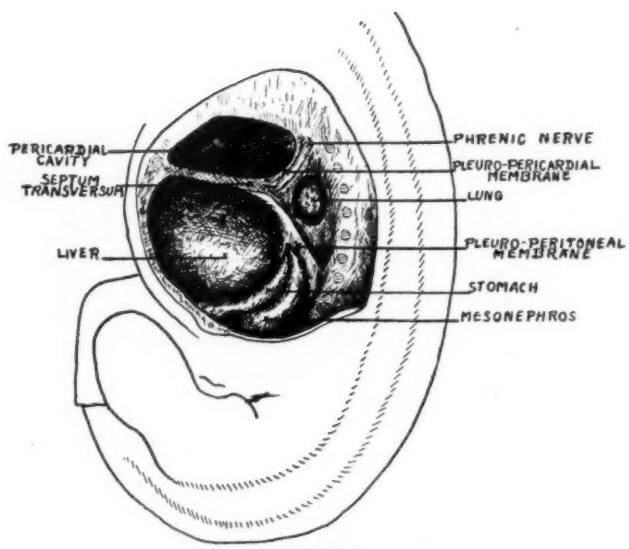
FIGURE 7.

Reconstruction of the 11 mm. embryo to show the relations of the pericardium, septum transversum, lungs and liver and their separation by the pleuropericardial and pleuroperitoneal membranes. (After Mall.)
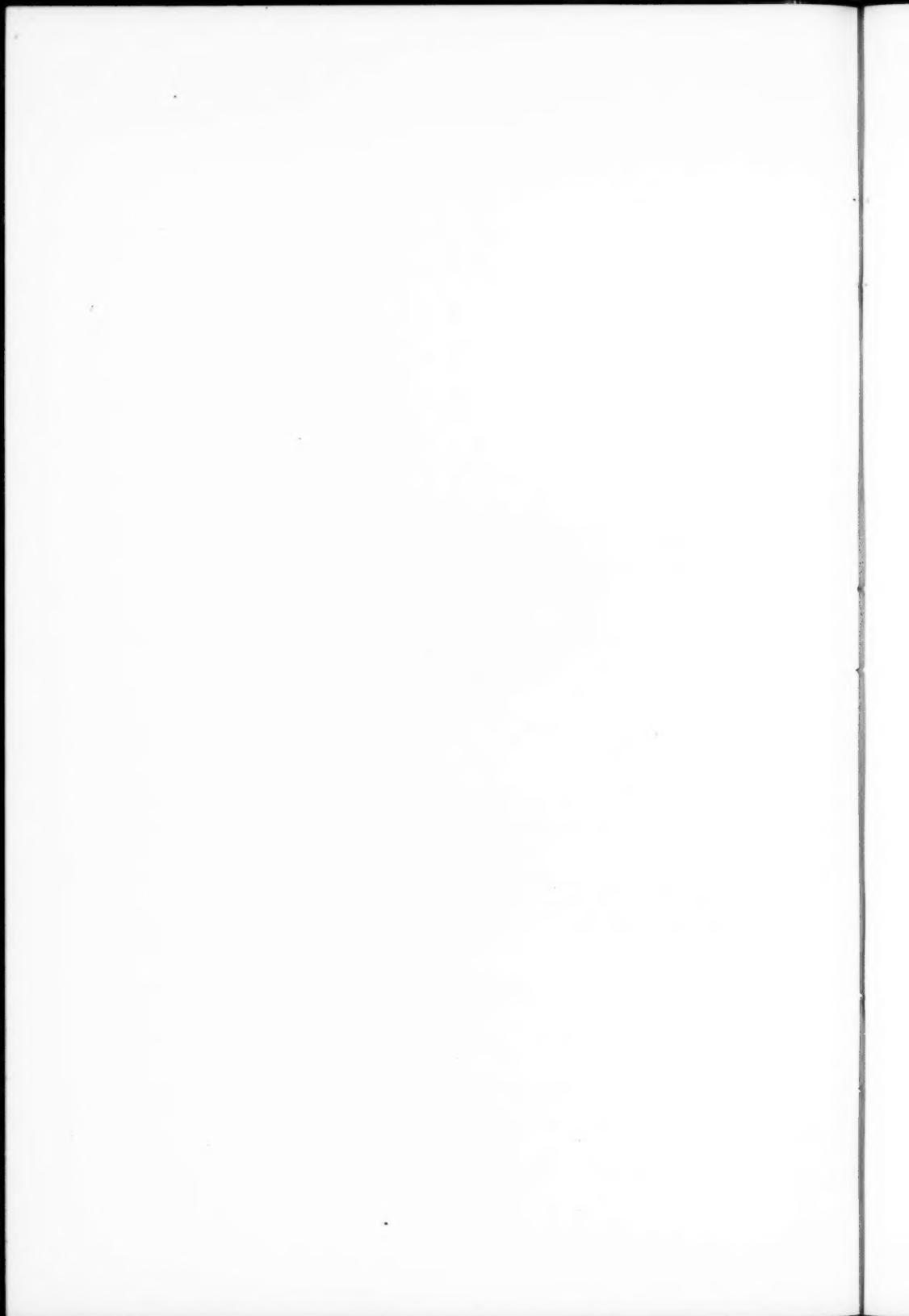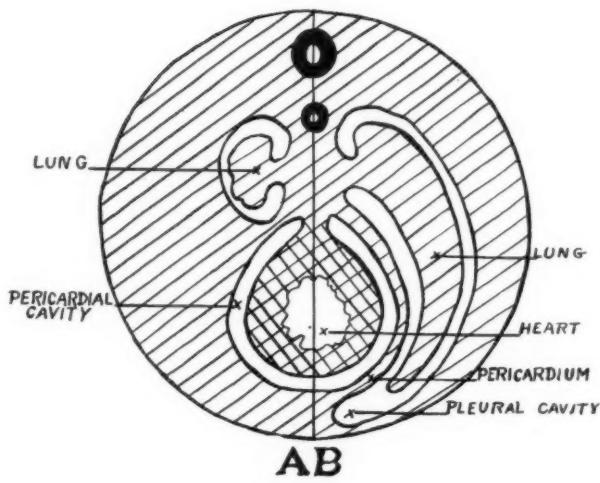
FIGURE 8.

Diagram to show the pericardium and its formation by the for-ward extension of the lung.

A.—Early stage.   B.—Later stage.   (Modified after Robinson and Prentiss.)
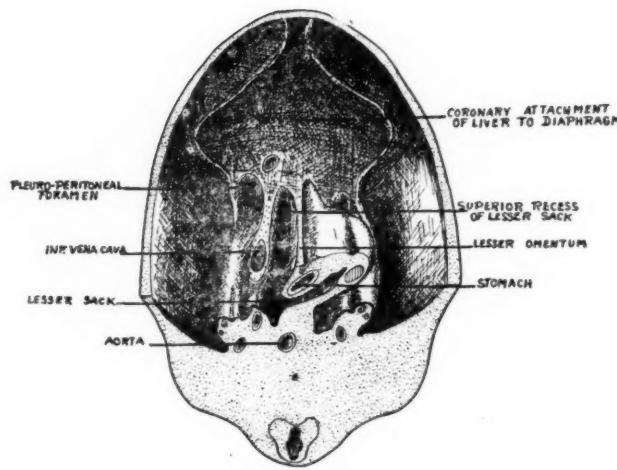
FIGURE 9.

Diagramatic ventral view of middle third of 12 mm. embryo. The figure shows the upward extension of the lesser sack parallel to the lower end of the esophagus. The liver has been removed from its attachment to the diaphragm. The communication between the pleural and peritoneal cavities via the pleuroperitoneal canals is still open. (Based on figures of Mall and F. T. Lewis, and model by H. C. Tracy.)
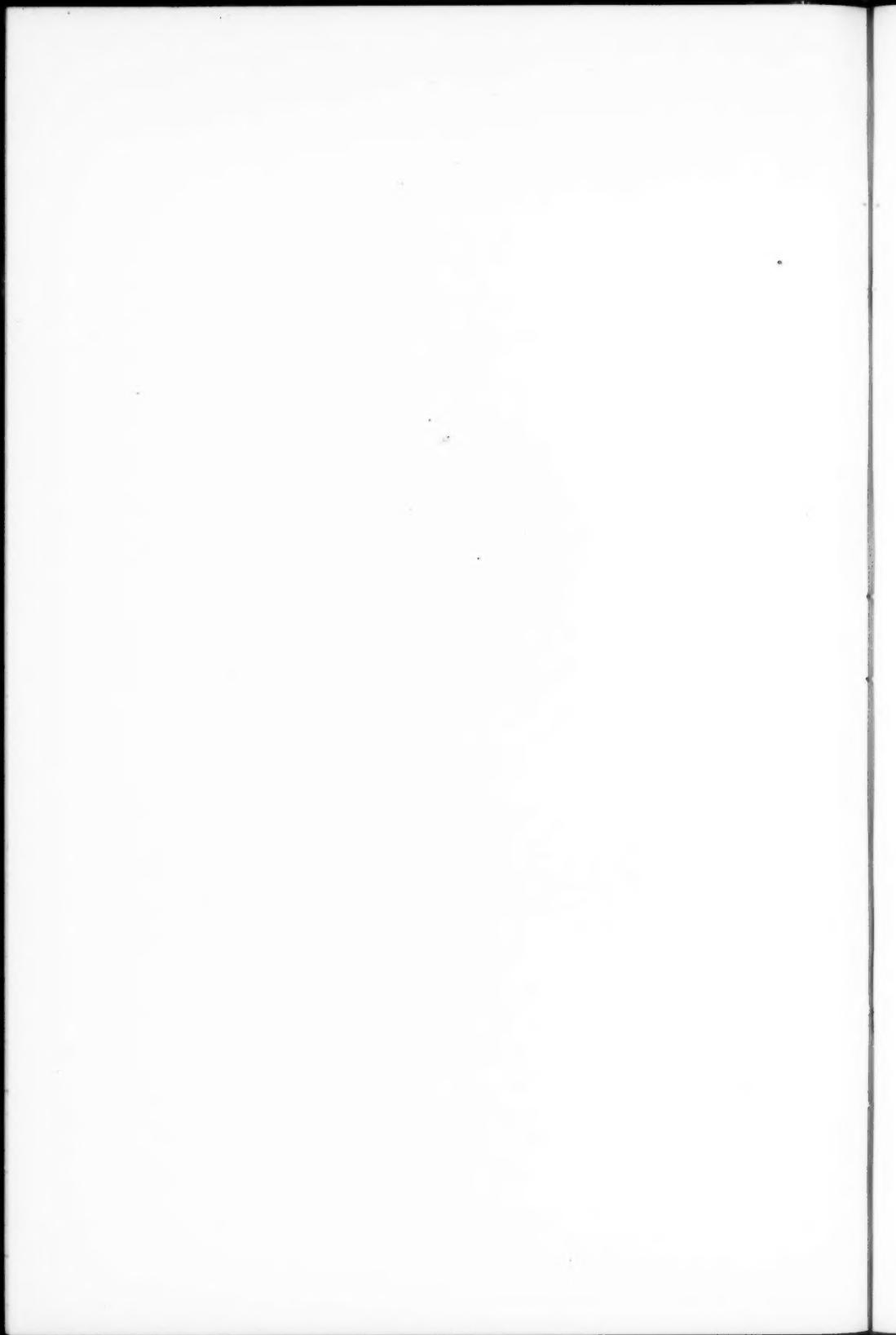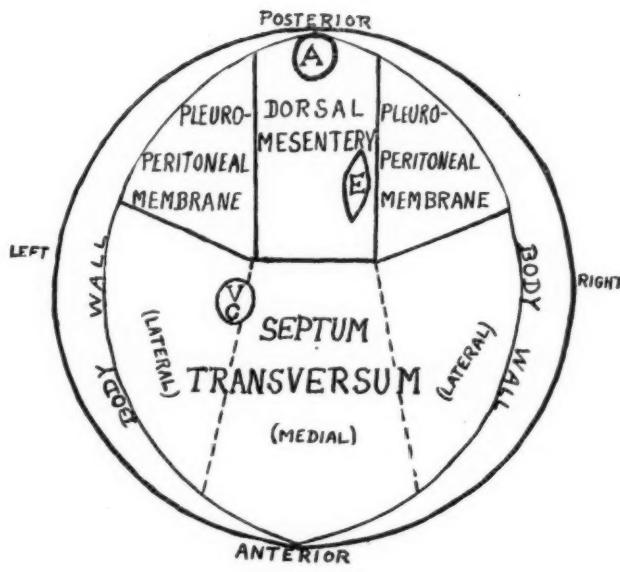
FIGURE 10.

Diagram to show the constituent portions of the adult diaphragm and the relative position occupied by them at an early embryological stage.

FIGURE 2.
Diagram to show the constituent portions of the adult diaphragm and the relative positions of each at an early embryological stage.
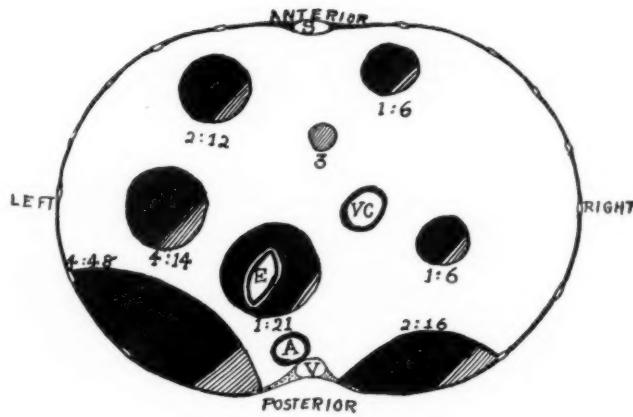
FIGURE 11.

Diagram to illustrate the positions and types of nontraumatic diaphragmatic hernia reported since 1900. The figures represent the number of cases in each location, the false hernias being shown in solid black, the true ones cross hatched.
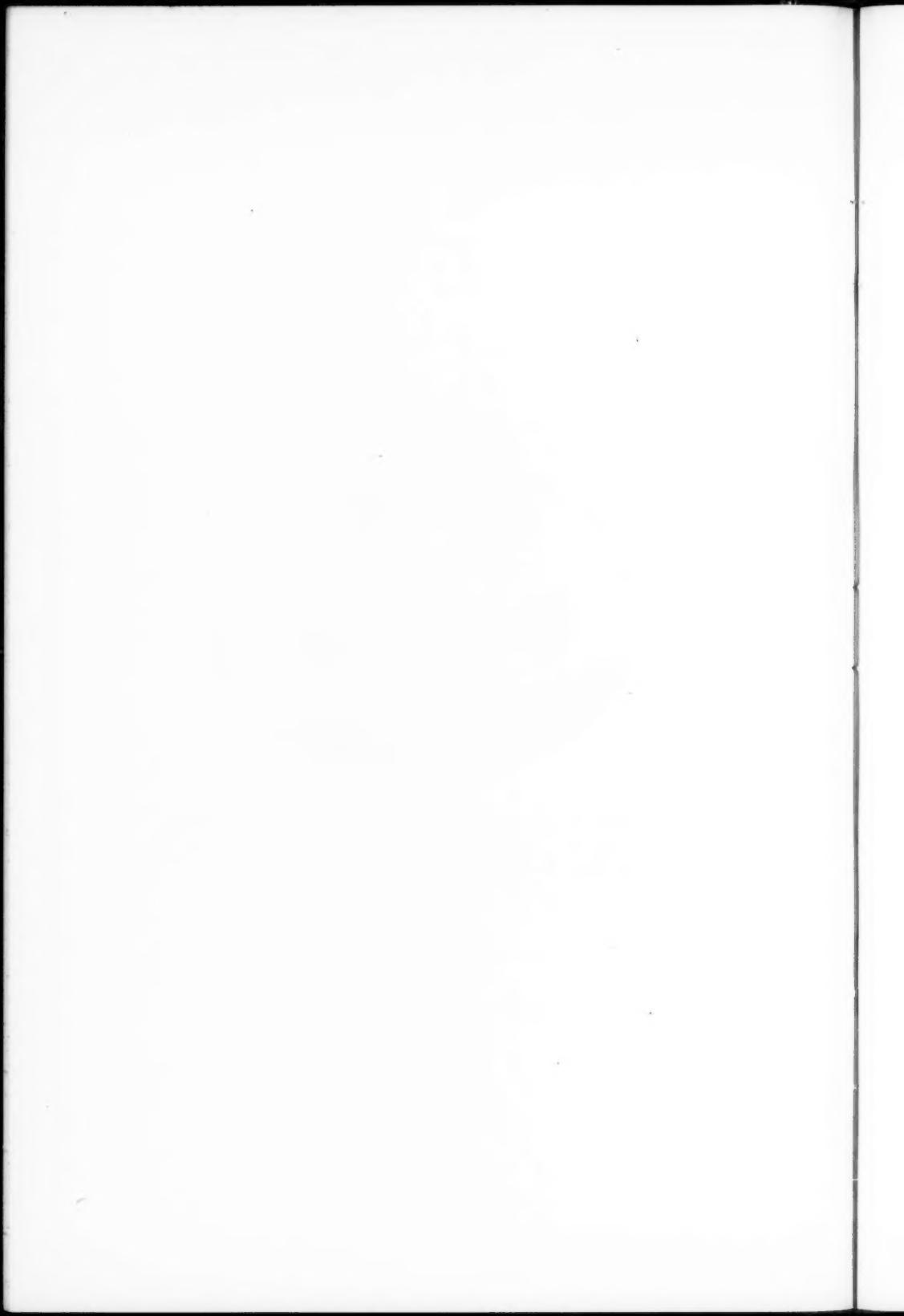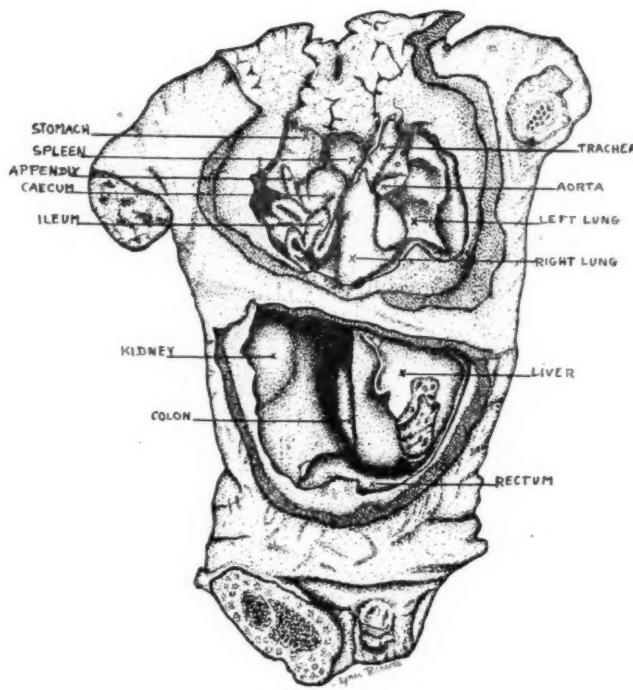
FIGURE 12.

Torso of male fetus, showing an extensive esophageal hernia of
the diaphragm. The stomach, spleen, small intestine, and a large
part of the large intestine have become lodged in the right thorax.
There is apparently no sack, an unusual condition in this type of
hernia. The heart has been removed, also the right half of the
liver, showing the relatively great size of the left half. (From the
collection of Dr. Harris P. Mosher, Harvard Medical School.)

LXXIX.

RADICAL OPERATIONS ON THE MAXILLARY SINUS AND DAMAGE TO THE TEETH.

By Robert H. Ivy, M. D., D. D. S.,

Philadelphia.

It has long been recognized that for the cure of the majority of cases of chronic inflammation of the maxillary sinus nothing short of a radical operation will suffice. It is conceded that nasal puncture and lavage will clear up most of the acute cases, but where the mucous membrane has become chronically thickened and the seat of polypoid degeneration, with or without bone necrosis, mere washing out through needle or canula from the nose has little effect. Again, this measure, while in the presence of free pus it generally establishes a diagnosis of maxillary sinus disease, may entirely fail to do so when the disease is manifested chiefly by the presence of solid granulation tissue. Establishment of free intranasal drainage by formation of a large window in the antranasal wall may occasionally be sufficient. However, in long standing cases, nothing is nearly so satisfactory for exact diagnosis and treatment as exposure of the interior of the sinus to direct inspection and palpation through a large opening from the mouth through the buccal wall. This permits removal of the diseased tissue with a curette, the operation being completed by establishment of permanent nasal drainage into the inferior meatus through a resection of the nasal wall and final closure of the original buccal opening. Objections have been advanced against these radical operations that they frequently result in permanent damage to healthy teeth in the vicinity of the buccal incision. Thus, J. L. Myers (Jour. A. M. A., 1921, lxxvii, 2081) calls attention to the possibility of devitalization of teeth by interference with their blood and nerve supply. At the suggestion of your Secretary I have endeavored to gather a few facts to determine whether or not there is any foundation for these objections.

Damage to sound teeth might conceivably be brought about in two or three ways: (1) By exposure of roots of teeth by removal of bone covering them; (2) By destruction of their vascular supply; (3) By destruction of their nerve supply.

1.—Exposure of Roots of Teeth by Removal of Bone Covering Them.—It is a generally known fact that if the supporting alveolar process and pericemental membrane about a tooth are destroyed by injury or disease, they will never be replaced, a focus of infection will usually be set up and the root will eventually be lost. It is conceivable that in doing the Caldwell-Luc or the Denker operation, if the incision through the buccal plate be made too low, the roots of some of the teeth near the apices might be exposed and thus converted into necrotic foreign bodies from loss of nourishing and supporting tissue. The nerve and blood supply of the pulp might also be cut off. Any such tooth injury would probably interfere with the success of the antrum operation, by setting up a new focus of infection. This accident is avoided by making the buccal incision well above the apices of the roots and being careful not to remove the bone too low down. None of the cases examined by me postoperatively showed either clinical or radiographic evidence that any injury to the teeth had occurred in this manner. Of course, these remarks do not apply to diseased teeth having a direct connection with the sinus prior to the operation, and which were removed at the operation.

2.—Damage by Destruction of Vascular or Nerve Supply of the Teeth or Both.—The arterial and nerve supply to the upper teeth, alveolar process and mucoperiosteum of the maxillary sinus reach these parts through minute canals in the substance of the bone and grooves in the wall and floor of the maxillary sinus. The posterior superior dental or alveolar artery and nerve enter the tuberosity of the maxilla on its posterior surface and supply more especially the molar teeth and surrounding alveolar process and gum, also the posterior part of the mucoperiosteum of the maxillary sinus. The canine and incisor teeth are supplied by the anterior dental artery and nerve which are given off in the infraorbital canal just before the infraorbital artery and nerve make their exit through the infraorbital foramen. They pass down canals in the anterior wall of the maxillary sinus to go to the teeth mentioned as

well as to the gums, alveolar process and mucoperiosteum of
the anterior part of the sinus.   The anterior and posterior
blood vessels form a very rich anastomosis in the floor of the
maxillary sinus, so that it is inconceivable that any individual
tooth could have its vitality impaired by cutting a dental
arterial branch unless immediately at the apex of the root of
the tooth.   There is usually a middle dental nerve branch
from the infraorbital canal which supplies more particularly
the region of the premolar teeth.   While the dental nerve
branches also form a very rich interlacing plexus in the floor
and outer wall of the maxillary sinus, there is, of course, no
true anastomosis of nerve fibers and the zones of distribution
are more clearly defined than in the case of the blood ves-
sels.   While some of the blood vessels must necessarily be
cut in radical operations on the maxillary sinus, the rich an-
astomosis will in all cases insure a sufficient collateral circula-
tion to maintain the blood supply of the tooth pulps.   Inter-
ference with blood supply by these operations, then, need not
be regarded as a factor in injury to the teeth.

Removal of a large portion of the outer bony and mem-
branous wall of the maxillary sinus of necessity produces a
resection of many of these nerve fibers to the teeth.   In the
Denker operation, where the bone removal is carried right
through to the anterior nares, a large section of the anterior
dental nerve is inevitably destroyed, with resultant loss of
sensation in the incisor and canine teeth.   Does mere loss of
sensation mean that the pulps of these teeth are devitalized?
I think not.   One can have anesthesia of the fingers due to
sensory nerve lesion, without gangrene.   Vitality depends on
presence of blood supply and not on sensory nerve supply.
Absence of sensation in a tooth does not necessarily mean that
the pulp is dead, although death of the pulp of a tooth follow-
ing inflammation is accompanied by loss of sensation.   Where
a tooth pulp dies from inflammation, decomposition will fre-
quently produce a change from the normal color, and such a
tooth will appear darker than its fellows with vital pulps.
These changes are not seen in teeth whose nerve supply has
been interfered with at such a distance that the blood supply
is not also cut off, as in fractures of the mandible or in radical
maxillary sinus operations.

A postoperative study of the upper teeth was made in a series of 20 cases which had been subjected to radical maxillary sinus operations, either Denker or Caldwell-Luc type, at periods ranging from three months to two years after the operation. As far as possible, it was determined by questioning the patient whether any teeth on the side of the jaw involved had been the seat of postoperative disturbance. Practically all patients noticed a numbness in the teeth and gums beneath the buccal incision, which lasted only a few weeks or months in some, in others for a year or more. The apparently sound teeth on the side of operation were then tested for pulp vitality by means of the faradic current. Where crowned or obviously pulpless teeth were present careful inquiry was made to determine whether devitalization had occurred prior to or after the antrum operation. When teeth did not respond to the faradic test or were obviously pulpless, radiographic films were made for evidence of periapical bone or root injury produced in the operation.

In the cases examined shortly after operation, the faradic test frequently showed absent or diminished reaction. This was particularly true in the canine and incisor teeth of cases subjected to the Denker operation, where the anterior dental nerve was removed. In practically all cases tested several months after operation absolutely normal faradic reaction was observed in the teeth, indicating regeneration of the lost segment of nerve.

In only one case was there any history of trouble in a tooth after the operation. In this case the lateral incisor had been treated for pericementitis due to a dead pulp, the radiograph showing an area of bone destruction about the apex. Even here it is by no means certain that this pulp was alive before the operation. In no case was there any radiographic evidence that injury to the periapical bone tissue or to the roots themselves had occurred at the time of operation.

While the series of cases is not very large, as a result of these examinations, I believe we are justified in concluding that fears of deleterious effects to the teeth from radical maxillary sinus operations of the Caldwell-Luc and Denker types, when performed by experienced surgeons, are entirely unfounded. Furthermore, if occasional damage to a tooth did

result, this should not be used as an argument to condemn a type of operation which is the only satisfactory curative procedure in many cases of chronic maxillary sinusitis.

Grateful acknowledgment is here made to Drs. R. H. Skillern, G. M. Coates, Ralph Butler, B. D. Parish, and others, whose cases in addition to those of the author furnished the data for this report.

SUMMARY OF CASES.

Operation.	Examination of Teeth.	Notes.
1. I. M. Sept. 21, '21. R. Caldwell-Luc.	Nov. 2, 1922. + + — m + — + 7 6 5 4 3 2 1	2nd premolar nonvital prior to operation. Pulp of lateral incisor probably became devitalized after operation, with area of periapical infection shown by X-ray.
2. C. B. Dec. 10, '21. R. Denker.	April 5, 1923. + + m + + + + 7 6 5 4 3 2 1	No abnormality found. Teeth felt numb for a time after operation, but this gradually passed off.
3. W. W. May 8, '22. L. Denker.	April 5, 1923. + + + + — m + 1 2 3 4 5 6 7	Normal sensation in all teeth except 2nd premolar, which was pulpless before operation.
4. E. B. Mar., 1920. R. Denker.	March 20, 1923. — m + + + + + 7 6 5 4 3 2 1	Sensation still slightly diminished in incisors and canine. 2nd molar nonvital before operation.
5. M. M. Nov., 1921. R. Side.	Nov. 1, 1922. m m m + m + + 7 6 5 4 3 2 1	Sensation the same as before operation.
6. M. M. Oct. 23, '22. L. Side.	Nov. 1, 1922. — — — + m m m 1 2 3 4 5 6 7	Numbness of inciscor and canine teeth one week after operation.
7. J. S. June 2, '22. R. Side.	Nov. 18, 1922. + m — — + + + 7 6 5 4 3 2 1	Diminished reaction in incisors and canine. Premolars devitalized before operation.
8. W. B. Mar., 1919. L. Side.	Nov. 18, 1923. + + + + m + + 1 2 3 4 5 6 7	Complete return of sensation.
9. C. B. May 13, '22. L. Caldwell-Luc.	Nov. 16, 1922. + + + + + + + 1 2 3 4 5 6 7	Complete return of sensation.

Operation.	Examination of Teeth.	Notes.
10. H. S. Dec. 10, '21. L. Denker.	March 22, 1923. + + + c m m c 1 2 3 4 5 6 7	Complete return of sensation. Crowned teeth normal.
11. L. F. Nov., 1922. L. Denker.	March 22, 1923. + + + + + + + 1 2 3 4 5 6 7	Complete return of normal sensation.
12. M. E. Feb., 1921. L. Side.	April 3, 1923. Bridge ⌒ + m — m c + + 1 2 3 4 5 6 7	Bridgework was on before operation. No change found, except slightly diminished sensation in central incisor.
13. E. G. Sept., 1922. L. Caldwell-Luc.	March 8, 1923. + — + + m + + 1 2 3 4 5 6 7	Negative reaction in lateral incissor, no X-ray abnormality.
14. E. G. Dec., 1922. L. Caldwell-Luc.	March 8, 1923. m m m + + + + 7 6 5 4 3 2 1	Still has numb feeling on right side, though teeth respond to current.
15. J. M. Dec., 1922. L. Caldwell-Luc.	March 28, 1923. + — + + + + + 7 6 5 4 3 2 1	Still has diminished reaction in 1st premolar. 1st molar devitalized before operation.
16. H. D. Nov. 1, '22. L. Caldwell-Luc.	March 23, 1923. + + + — + m + 1 2 3 4 5 6 7	Numbness gradually passing off, except in 1st premolar. No other abnormality found in this tooth.
17. S. D. Jan., 1923. R. Caldwell-Luc.	April 7, 1923. + + — + — — — 7 6 5 4 3 2 1	All teeth responded to test before operation. Sensation has not yet returned to certain teeth, but they show no other abnormalities.
18. J. G. Jan., 1923. L. Side.	April 9, 1923. + — + + — + + 1 2 3 4 5 6 7	Numbness still present in lateral incisor and 2nd premolar.
19. F. B. Feb. 8, '23. L. Denker.	April 6, 1923. + m m m m m 1 2 3 4 5 6 7	Sensation normal in central incisor.
20. W. N. Feb., 1922. R. Denker.	April 21, 1923. c m m m c + + 7 6 5 4 3 2 1	Complete return of sensation in incisors. Canine and 2nd molar now used to support bridge.

Explanation of notation: 1, central incisor; 2, lateral incisor; 3, canine; 4, 1st premolar; 5, 2nd premolar; 6, 1st molar; 7, 2nd molar; plus sign, presence of sensation; minus sign, absence of sensation; c, crown; m, missing.

LXXX.

OTITIC CHOLESTEATOMATA.*

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It is not my purpose to review extensively the literature of otitic cholesteatoma, my chief object being to present an unusual case of extradural tumor of this nature; neither shall I consider the various well recognized methods, other than surgical, of treating aural suppuration, whether cholesteatoma be present or not. The object of this paper is to bring forth a discussion of the etiology, histology and pathology of otitic cholesteatoma, its surgical removal and the prevention of its recurrence in so far as possible. It is interesting to note that the more recent views differ but little from those of the masters of twenty or thirty years ago; indeed, the whole present conception of this peculiar formation is based on the contributions of an earlier period.

With the object of determining whether cholesteatomata are found in osseous structures other than the temporal bone, their legitimate and historic habitat, I have inquired of a number of general surgons, including J. Chalmers DaCosta, John B. Deaver and Charles H. Frazier, all of whom gave a negative answer, the notable exceptions being Harvey Cushing[1] and Percival Bailey,[2] but the tumors which they report may differ, however, in some important aspects from the otitic cholesteatomata long since recognized by aurists as a consequence of chronic otorrhea. We all recognize the migratory propensity of cholesteatomata and their ability to destroy the soft tissue and penetrate the osseous structure; it would not be unreasonable, therefore, to assume that growths of this character adjacent to the organ of hearing had their origin in the temporal bone, even though no active process in the latter was immediately discoverable.

Cases are reported in which cholesteatomata have been found in the eye, brain, spinal cord and kidneys. It is possible also that these might differ in some essential respects from the type under consideration.

Should an aural discharge, however innocuous, continue for a long period, necrotic destruction of the connective tissue elements ensues. In the presence of a diminishing suppuration, new connective tissue formations are induced, which serve to protect the underlying osseous structure, so that it frequently resists the ravages of the various microorganisms and the necrotic changes incident thereto for an indefinite time. Sooner or later, however, the normal blood supply and the nutrient vessels are interfered with, and those parts least supplied, such as the incus, first succumb.

Once the soft parts have been destroyed, the tympanic and its accessory cavities are wholly deprived of their every means of resistance to the devastation of invading bacteria, and thus originate caries and necrosis and the nidus for the development of cholesteatoma.

When the pneumatic cells of the mastoid process become obliterated in the course of a chronic proliferative osteitis, acute exacerbations are more conducive to intracranial complications through caries of the tympanic roof or the posterior wall of the mastoid, exposing the dura or lateral sinus.

Gruber and other early authors believed these cholesteatomatous formations to be malignant in character, and seemingly substantiated their conclusions by minute examinations of the masses. More recent views consider them primarily benign, and if ever malignant they become so in the evolution through which they pass. It is also shown that these masses invade the osseous structure through the Haversian canals, even in the early stages of erosion and before caries and necrosis supervene, which explains the tendency to recurrence after thorough extirptaion seems to have been attained.

These masses, composed of concentrically arranged epithelium, cholesterin crystals and inflammatory débris, literally swarm with microorganisms. Their gradual growth or enlargement first destroys the soft parts and then produces dilatation of the bony cavities, more especially the mastoid antrum, and this in turn is followed by pressure necrosis of the under-

lying osseous structure, frequently exposing and subsequently involving the dura, lateral sinus, and rarely even the brain itself in an acute process of extremely dangerous potentiality.

Cholesteatomata vary in size from that of a small pea to a walnut. They are usually round, but their final contour is governed somewhat by the shape of the cavity in which they form; however, when the bone undergoes necrotic change the mass may penetrate it and mould its outline according to the resistance encountered.

Except for occasional mild attacks of giddiness and headache and a feeling of weight and pressure in the head, cholesteatomata may remain in the temporal bone for years without causing symptoms. Curiously enough, cases of this kind do not seem to be recognized as of sufficient importance to interest the patient and sometimes not even the physician. Several of my recent cases did not seek relief until they had had severe attacks of vertigo, even to the extent of necessitating an attendant. Others suddenly became almost totally deaf or developed Bell's palsy before they were impressed with the gravity of the situation. Although these patients are aware of the existence of a chronic ear disease, a fair percentage insist that they cannot remember how long ago an actual discharge was present, this in spite of the fact that an examination reveals an otorrhea, frequently very offensive.

Under the stimulus of an exacerbation, the mass, wherever situated, takes on acute inflammatory changes which lead to extensive destruction of tissue through necrosis. In their development, cholesteatomata of the tympanic cavity may follow the line of least resistance and extend well into the external auditory canal; on the other hand, cases have been reported as penetrating the eustachian tube. The latter I have never observed, but I have seen them through a carious erosion of the mastoid cortex. Likewise, they may cause labyrinthitis, meningitis, sinus thrombosis or brain abscess.

A unique case which I have hitherto hesitated to report, but which will be detailed later on, deals with a large cholesteatoma which invaded the tempral lobe, being thoroughly walled off and enclosed in a capsule, perhaps spurious in type, the patient presenting no symptoms, either local or general, indicating its presence.

I have never seen what may be termed true cholesteatoma except that which developed in the course of or as a result of chronic otitic suppuration, and, furthermore, my observation leads me to believe that the tympanic and accessory cavities, together with the marginal perforations of the membrana tympani, are alone the site of these strange formations. Some are reported occupying the external auditory canal, but when it is remembered that the meatal epidermis does not proliferate pathologically, and thus give rise to the pearly white dermic scales and other characteristics until it reaches the tympanum and adjacent cavities, it is reasonable to assume that these recorded meatal cases are not of the true cholesteatomatous type. Formations of cholesteatoma are, of course, found in the deep external canal, but are associated with and are actually an outgrowth from the process in the middle ear cavity, or protrude through a fistula leading into the attic or antrum.

Those who have seen a considerable number of cholesteatomatous cases must have been impressed with the following facts: That they are observed only in the presence of chronic suppuration; that their formation is inhibited when the otorrhea is profuse; that they are practically never found in central perforations of the membrana tympani unless the margin is partially adherent to the tympanic wall; and that their development nearly always occurs in chronic suppuration of the attic or perforations of the superior and posterior quadrant. This would seem to support the generally accepted theory that the primary stage of their evolution is the ingrowth of epithelium through the marginal perforations; their subsequent well known characteristics being developed by the histologic changes through which they pass.

Another distinctive feature of cholesteatoma is the temporary decrease of suppuration, almost to the point of cessation in some instances, when the mass has attained a considerable size. This is the type of case that may be quiescent until some moisture or infection invades the ear, producing recurrent attacks of mastoiditis, when they swell hygroscopically, as is shown by headache, vomiting, vertigo, facial palsy, etc., ushering in the well recognized and dangerous acute exacerbations. It is in this recurrent type of case that I have observed the great majority of intracranial complications.

Lucien-Barajas[3] has clearly shown that there are two formations found in the middle ear under the name of cholesteatoma. One is composed of granular, fatty and purulent masses of detritus, intermixed with squamous epithelial cells, keratin and crystals of cholesterin, while the other variety is formed by imbricated nodules concentrically arranged like the layers of an onion. These peculiar nodules consist microscopically of large fat, endothelial cells, polygonal in shape, the nuclei staining but faintly, while the masses contain a large quantity of cholesterin. The major portion of our cases belong undoubtedly to the first group and must not be classed as true cholesteatoma, nor should similar masses be placed in this class when composed of polystratifications of keratinic epithelial cells, which develop inflammatory dermal affections of the external auditory canal and which are horny or keratinic formations.

Personally I doubt the existence of the socalled primary otitic cholesteatomata. To be such they must necessarily originate independently of tympanic suppuration, and therefore not pass through the characteristic evolution. This would mean either that they are not cholesteatomata or else the tympanic perforation may have temporarily closed over the underlying mass, of which this tumor was originally an outgrowth. Ballenger,[4] speaking of secondary cholesteatoma, says this is the type found in practice ; the primary form is chiefly limited to literature.

Meatal epidermal layers may find their way into the tympanic cavity during the course of a receding chronic suppuration, and thus provide the necessary epidermal lining to produce the socalled spontaneous cure of a chronic suppurative otitis media. It will be seen, therefore, that the mere presence of epidermal scales in a chronic middle ear suppuration does not in any sense constitute or portray the distinctive features of cholesteatoma. Epidermal layers are an integral part and essential to ultimate completeness of the finished product, but this end is not accomplished until the underlying osseous structure becomes involved, and the perforating epidermis degenerates into excessive pathologic proliferation and the course of evolution through which it passes is completed.

To some extent hyperplasia of the epithelium occurs in all cases of chronic otitic suppuration, but these formations cannot acquire the typical cholesteatomatous state until they are excessive and have remained amid the environment of the middle ear, attic or antrum sufficiently long to pass through the distinctive cholesteatomatous evolution.

The admittance of moisture in some instances causes decomposition of the mass, which is followed by the formation of granulation tissue or even polypoid growths, and this in turn favors the spreading of septic material to the meninges, brain and blood vessels through the osseous deficiencies incident to the changes resulting from pressure necrosis, the extent of which is dependent almost wholly on the size of the ever increasing mass.

Granulation tissue or polypoid growths originating in Shrapnell's membrane, more particularly in the superior posterior quadrant, are, in my experience, almost always the outgrowth of cholesteatomatous masses occupying the attic and sometimes the antrum also. When the lower part of the membrana tympani is intact, the characteristic sound produced by inflation is not heard, owing to the fact that the mass in the upper part of the cavity seals off the opening in Shrapnell's membrane. In such instances it is not uncommon for the incus, the head of the malleus and their ligaments to be absorbed through pressure necrosis.

Pressure atrophy and consequent necrosis due to cholesteatoma have been the principal underlying factors in virtually all of the cases of the socalled nature's radical mastoid operation that I have observed. On the other hand, I recall several patients in whom the cavities of the antrum and middle ear were wholly exposed, through nature's operation, and yet were both dry and empty.

Cholesteatomata not only offer a most favorable breeding ground for the rapid multiplication of organisms, but the very nature of their component parts serves to protect the germs and the new pabulum as it is secreted against our efforts at extinction by means of antiseptics, germicides and so on. Our chief reliance, therefore, as an initial step for relief lies in surgical intervention.

The cholesteatoma described by Virchow, a heteroplastic new growth of congenital origin, must be differentiated from that long since recognized by aurists, which is always secondary to a chronic otorrhea. I doubt very much whether a suppurative otitis media is ever caused primarily by cholesteatoma, as intimated by Harvey Cushing;[5] if so, then our conceptions of the etiology and histology of otitic cholesteatoma must undergo complete revision.

I have read with much interest the informative contributions of Harvey Cushing[6] and Percival Bailey[7] referring to extradural cholesteatomata. After reviewing several cases by various authors, Cushing states that "these cases constitute a group of true epidermal cholesteatomata which are extradural and appear to originate between the two tables of the skull. The tables become separated, thinned by pressure and finally absorbed, the inner table as a rule suffering more than the outer. . . . Unquestionably the removal of the epidermal membrane is the crux of the procedure, and this can only be accomplished before the cyst is emptied of its contents and its enveloping membrane permitted to collapse. Incomplete removal of the lining wall is doubtless the explanation of the recurrences so common after operation by otologists for the cholesteatomata of the temporal bone which come almost solely into their province."

From the fact that otitic cholesteatoma does not usually develop until the soft parts have been mostly destroyed and the bone involved at least in a simple erosion, it is evident that aurists do not encounter the distinctive "lining wall" referred to by Cushing, and furthermore, it seems to be generally conceded that recurrences in otitic cases, to which he alludes, are the result of a subsequent outgrowth of the mass which has penetrated the Haversian canals and not to incomplete removal of an assumed "lining wall."

In operating, it is of course our object to remove as thoroughly as possible all new connective tissue, as well as other inflammatory débris, and I feel that we accomplish our object reasonably well, especially when due consideration is given to the dangers and difficulties encountered in the field of operation, which includes the thin plates of bone covering the fallopian canal conveying the tympanic branch of the facial nerve,

the carotid artery, the jugular bulb, various parts of the labyrinth and the lateral sinus.

Bailey[8] intimates that Cruveilhier's "pearly tumors" are never found in the temporal bone complicating a chronic suppurative otitis media.   It may be true that otitic cholesteatomata do not contain all of the characteristics of this classic type, as demonstrated by laboratory findings, and yet from a clinical standpoint some answer to this description in almost every detail.   After all, may it not be a reasonable assumption that these extradural cholesteatomata had their origin in a former otitic disease of the type that assumed a spontaneous cure? If otology is to be deprived of the distinctive position it has maintained relative to the etiology and histology of otitic cholesteatomata, it will at least require much additional research and clinical observation before surrender of our seemingly well established facts will be acknowledged.

In order to secure permanent relief, cholesteatomata must be removed in their entirety so far as this is possible.   Even then, if the suppuration continues, the irritation resulting is apt to cause recurrence, and when the suppuration ceases continued desquamation and proliferation are very apt to bring about the same result.   It is evident, therefore, that the two objects to be attained after removal of the mass are cessation of the discharge and cessation or limitation of pathologic proliferation.   I believe that we have at our command corrective agencies to accomplish both of these results in a favorable percentage of cases.   Skin grafting, especially as practiced by Dench, after Ballance, seems to give very satisfactory results. For those of us who are not so skilled in this procedure, packing the cavity with small strips of gauze thoroughly moistened with a 1/1000 or 1/500 solution of acriflavin (the former strength being mostly used), will prevent the recurrence of cholesteatomata in many cases, even of a most obstinate type. This packing must be renewed at first every day, then every third or fourth day, until all evidence of further discharge or proliferation has ceased.   Should there be evidence of recurrence afterwards, I have found a liberal application of a 1 per cent or stronger solution of mercurochrome to be quite efficacious—in fact, at this stage mercurochrome gives even better results than a continuation of the acriflavin.

After all, we must consider the complete eradication of cholesteatoma as not only difficult, but in some cases perhaps impossible of accomplishment, from the fact that even in slightly eroded bone the mass will force its way into the Haversian canals, forming focal centers from which it may develop in the future.

I have been using acriflavin for about three years in the treatment of cholesteatoma and mercurochrome for a somewhat shorter time, and although sufficient time has not elapsed for me to make a definite statement as to the permanent benefit thus derived, this treatment does seem not only to inhibit the formation of cholesteatoma, but acts as a specific against recurrence after thorough removal.

A sane rule to follow is that when a diagnosis of cholesteatoma has been made, or even when there is a strong probability of its existence, surgical intervention should be recommended if the case does not yield promptly to nonsurgical treatment.

When there is sufficient reason to believe that the process is limited to the tympanic cavity, operation through the external canal is justified. This operation necessarily includes removal of any granulation tissue and polypi that so frequently accompany cholesteatoma, as well as excision of the fragments of the membrana tympani, malleus and incus. Following removal of the mass, and ossiculectomy, packing with acriflavin solution, 1/1000, followed by the use of mercurochrome, as above described, should be carried out carefully to correct the otorrhea and prevent the return of pathologic proliferation.

The report of the case I referred to above follows:

J. W., male, aged 33 years, locomotive engineer. Well nourished, apparently enjoying best of health, very alert mind. Good habits and denied venereal infection. Suppurative otitis media, recurrent in type, extending over a number of years. Six years ago otorrhea ceased for four years, although patient admitted "a slight moisture in the ear" at times in the interval. Two years prior to operation an acute exacerbation followed the entrance of water into the ear, causing earache, pain in head, vertigo and emesis for a brief time. Discharge of varying quantity was constant from this date until relieved by operation. Latterly the discharge was scanty, brownish yel-

low, blood streaked and offensive. Patient stated that his ear was "punctured," whatever this procedure may have been, at onset of exacerbation, and the discharge continued to be sanguinolent during the intervening two years. Patient was free from all general symptoms but sought relief from an annoying discharge.

Left ear normal. Membrana tympani of right ear largely destroyed. Granulation tissue and what seemed to be a cholesteatomatous mass protruded through a large opening in the superior posterior quadrant and the necrotic opening in the adjacent posterior osseous wall. No ossicles observable. Nothing abnormal revealed by ocular examination. Chest normal with the exception of a few rales, the result of a recent cold. Abdominal examination negative. Blood examination and urinalysis normal.

A radical mastoid operation was performed. Cortex extremely hard and thick. Cells below level of antrum, having undergone sclerotic changes, were filled in with solid ivory-like bone. Antrum of normal size, filled with a cholesteatomatous mass, some granulation tissue and a very little free pus. Through the destroyed tegmen antri and tympani this cholesteatomatous mass pushed its way into the interior of the skull, forcing the dura before it through the temporal region to a depth of about 1¾ inches.

Before reading Cushing's instructive monograph[9], wherein he reports a cholesteatoma the size of an adult fist, weighing 175 grams, four inches in diameter, and not producing any general symptoms indicative of its presence, I hesitated to report this case, as I could not conceive that the dura could be reflected to such an extent without causing some symptoms.

The opening through the tegmen antri and tympani was much smaller than the cavity beyond, so that the mass ballooned considerably at its distal boundary. This cholesteatomatous mass was first removed from the antrum and then from the ballooned intracranial cavity. On account of the possibility of inducing a meningitis in a healthy, ambulatory case, no attempt was made to remove the growth en masse, so I can only say that the tumor approximated in size a small pear. It had every characteristic of a typical otitic cholesteatoma.

The walls of the resulting cavity pulsated noticeably on removal of the mass.

The cavity was packed with a 1/1000 acriflavin solution, this being changed every day. The patient made an uneventful recovery and is now following his usual occupation.

As this was the first growth of this type that I had ever seen involving the interior of the skull, I asked my colleague, Dr. J. Clarence Keeler, who assisted in the operation, to furnish me his own estimate of its position and size. This he gave me as follows:

"The cavity, which was filled with cholesteatoma, appeared to be in the brain tissue, walled off by a thin layer of connective tissue. The walls pulsated after the cavity was evacuated. Location and size of cavity measured by memory on an adult temporal bone: Cavity was above the tentorium and extended along the anterior bar of the petrous bone to the extent of $1\frac{1}{2}$ inches. About 5/16 of an inch of the brain cortex formed the outer wall lying next to the internal surface of the squamous plate, which did not appear to be involved. The upper depth of the cavity was about $\frac{3}{4}$ of an inch. The cavity extended upward, forward and inward as given per measurements, with a width of about $\frac{5}{8}$ of an inch."

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LXXXI.

A SURVEY OF THE HAY FEVER QUESTION—A CRITICAL REVIEW OF THE SITUATION RELATIVE TO THE ETIOLOGY AND TREATMENT.*

By Otto J. Stein, M. D.,

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In order to discuss certain phases of the hay fever question it will be necessary to make at least a cursory survey of the subject in general, including, with the seasonal pollen disease, several of the vasomotor types of rhinitis.

The name "hay fever" has frequently been applied to most any form of vasomotor disturbance of the nose, although improperly so. The term itself does not correctly express the full meaning of the disorder. The acceptance of pollen causation should firmly establish its nomenclature, and the name pollen disease, or perhaps pollenosis as suggested by Todd, should preferably be used. Although Walker still speaks of a "perennial hay fever due to animal emanations and foods." (Jour. A. M. A., September 18, 1820, and Annals of Otology, Rhinology and Laryngology, September, 1922.)

In the past hundred years many theories have been advanced as to the cause, and as many different names have been used. Beschorner in 1886 found in the literature fifty or more names for the disorder. This was because the nasal symptoms of pollen disease are the same as those of a multiplicity of other causes. Many of the theories that were then advanced are inapplicable to the present conception of this disorder. Some of them are true pollen disease of seasonal character and respond to the cutaneous test, while others are a type of hyperesthetic rhinitis of diverse etiology, sometimes being mechanical, bacterial or of fish, fowl or animal origin. A logical no-

menclature would be pollen disease due to pollen causation and hyperesthetic rhinitis, a vasomotor nasal disturbance of manifold etiology.

From the time Bostock gave the first concise description of the disorder in 1819, under the name catarrhus aestivus, down to Dunbar's publications in 1902 and 1903, many theories prevailed as to the cause. These various causes were supposed to affect the nose: First, from without, as from solar heat rays, light rays, fine dust, odors, essential oils, chemical fumes and animal substance. Second, from within the nose, as synechia, rhinolith, septal ridge and spur, hyperesthetic areas and polyps. This view was sponsored by Daly in 1881 and enthusiastically supported by Roe, Sajous, Hack, Hertzog, Bosworth and others. Third, from a perverted state of the body fluids, chiefly an excess of uric acid in the blood creating a condition called uricemia. The champions of this theory were Molliere, 1884; Leflaine, 1887; Cheatham, 1890; Leseur, 1895; Haig and Bishop in 1894.

The theory of neurosis and psychic disorder was stoutly supported by John C. Mackenzie and followers in 1884. Some went so far as to class them ironically either as "neurotic, erotic or tommyrotic." After this came the theory of infection from Helmholz, Heyman and Sticker. In 1900 Holbrook Curtis enthusiastically sponsored a preparation made from common ragweed, under the name of liquor ambrosia, while Ingals experimented with a liquid made from ragweed and goldenrod.

For a time interest in the subject lagged, then suddenly Dunbar created local interest by the presentation of his extensive investigations, wherein an albuminous principle obtained from pollen of certain cereals and grasses was isolated and to which was ascribed toxic action. At first only passive concern was aroused, even though Elliotson in 1831, Majendie in 1839, Abbott-Smith in 1865 and Blackley in 1872 had presented ideas somewhat similar. They ascribed the irritation of the pollen to a mechanical action of its spines and prickles and not to a protein of a toxalbuminous nature as Dunbar did. This toxic principle, extracted from wheat and rye and later goldenrod, was injected into young horses, after trying goats and rabbits, and he obtained a serum of antitoxic action. Ow-

ing to its unstable nature, it was mixed with an inert powder and sold under the name of pollantin. MacCoy referred to this as the first instance of the production of antitoxic serum where the animal and vegetable product have been crossed. He obtained complete and prompt relief in fifteen cases. Somers made a serum from animals inoculated with goldenrod pollen and reported prompt and positive amelioration of symptoms in a majority of ten cases with a smaller number of complete disappearance. Mohr, Joachim, Loeb, Stein, Semon, Curtis and many others used it with varying results, in the main successful as to the amelioration of symptoms and a shortened time. Its disuse was occasioned partly because of the necessity for a special serum from each pollen cause. After Dunbar awakened interest in the theory of toxicity from plant pollens and also in his passive immunity with serums, attention was directed to other proteins, such as animal hair and skin, feathers of birds and the proteins from foods and bacteria. Clark and Meyer in a recent article report a unique case of susceptibility to silk from the glue used in its manufacture. The result of these numerous observations brought forth a long list of new causes added to the list of known vegetable like pollens, so that the determination of the particular offending protein often assumes the proportion of a huge task. In order to learn the nature of an individual's susceptibility, a skin test may be made, using such proteins as may suggest themselves from a careful history of a given case. A certain amount of skill is necessary in its performance. At least care in its performance and caution in its interpretation are demanded. Skin tests may be misleading and not infrequently reveal multiple causes. The younger the age, the more sensitive the test. Where the cause, for instance, is found to be a food, or a toilet article, or wearing apparel, its elimination may suffice in bringing relief. A change in geographic locality may remove the offending pollen. These are simple instances of simplified treatment. After having determined the offending protein by allergic reaction or otherwise, the next natural step was to prepare a vaccine that would desensitize the individual. As has been the case in the past with other forms of treatment, much enthusiasm has been aroused. Many able and conscientious workers have contrib-

uted to the large volume of literature on the subject, and distinct progress is noted of late in improved technic. Conspicuous in this field of endeavor are the names of Noon and Freeman in 1911, who were pioneers in demonstrating active immunity, Vaughan, Abderhalden, Clowes, Koessler, Goettlieb, Coco, George Mackenzie and Walker. Their writings summed up indicate that in applying treatment for active immunization particular attention should be given to the following points: 1. A careful study of each case to ascertain its specific protein or proteins. 2. All contributing causes, constitutional or local, must be corrected. 3. The proper preparation and preservation of the vaccine. 4. The time of commencing treatment. This must be early enough to permit the development of a gradual immunity, about three months prior to the expected attack. 5. The time to stop treatment. Even though preseasonal treatment has been taken, severe anaphylactic shock may result from a sudden exhibition of quantities of protein, such as may occur at the height of the flowering season. Garnet (Jour. A. M. A., September, 1920) anticipates this by applying adrenalin to the nose at this time. In the case of food proteins it may also be necessary to withdraw the faulty food. 6. Exact dosage, commencing with minimum strength and amount and very gradually increasing. According to Walker (ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, September, 1922), a 0.1 or 0.2 c. c. of that strength solution should at first be used, which just failed to give any skin reaction. 7. Interval between treatments. Usually five to seven days.

Failure to observe these general rules, through ignorance or carelessness, may result in serious, if not fatal, consequences. Bessau says: "Active immunization against hay fever is not entirely free from harm and must be practiced with greater caution." He reports a case of a female, 20 years old, who, after the seventh vaccination in fifteen months, complained of a feeling of contraction within the nose, face and throat; sneezing, vertigo, great weakness and prostration. Cyanosis, edema, particularly of the nose and ears, dyspnea, mucus in the air passages, weak compressible pulse, cold hands. After two hours, urticarial rash, towards evening many symptoms still present. During the night very restless, mouth

breathing and an attack of sneezing after midnight, twenty odd times, with lacrimation, itching and so forth, typical hay fever symptoms, which disappeared in half an hour. During the following day some of the symptoms still persisted. Dunbar, Walters and others have recorded similar experiences with pollen poisoning. Ersner raises the question whether hay fever and asthma are due to a deficient protective substance in the body or the result of overstimulation with toxins. He believes overstimulation is the answer, and to administer pollen during the acute stage of hay fever is adding a torch to the fire already burning. Patients thus treated become worse, and no pollen treatment, therefore, should be given at this time. He tries to develop an immunity by beginning his injections at least three months before the time of the attack, and then starts the injection of stock vaccine against the rhinitis that accompanies the hay fever. Most of the authorities (Gottlieb, Sheppegrell, Selfridge and Walker) insist on the individual pollen treatment determined by previous skin tests. Others still use mixed pollens. If of the same biologic type this would seem permissible, although evidence has shown otherwise at times. To carry out a consistent pollen treatment, recognition must be given to those pollens in a given geographic area, even if immunity is obtained from related botanical groups (Goodale). No immunity results from unrelated groups (Clowes). Hence the treatment of the East is not necessarily the remedy for the West, and this is also true of the North and the South, Europe and America.

There is an economic side to this question that is never commented on by the physician, but very much so by the patient, namely, the cost of the treatment in time consumed and services rendered.

The pathology of bacterial disease causing hyperesthetic nasal symptoms is generally understood and accepted. The pathogenicity of pollen disease is less understood. In fact, it seems to be the weakest link in the chain. There must be an altered state of the chemistry of the body fluids which creates the individual's susceptibility. That here lies the fundamental cause of the disorder only a heretic can deny, and much hope is placed in the investigation that physiologic chemistry must soon reveal. Majendie (1839) and later Wolff-Eisner

suggested a protein sensitization by parenteral digestion as the underlying cause of the reaction. Koessler (Journal of Infectious Diseases, 1914) emphasizes this when he says "the proteolytic enzyme of the nasal secretions splits the foreign pollen gradually into harmless proteoses and aminoacids," and the small amount of contained toxin is so slowly and in such minute amounts absorbed as to be negligible in effect. A sudden and large amount of toxin resorption injures the epithelial cells, so as to permit easy permeability of the proteins. There also results a local tissue sensitization through the production of a proteolytic ferment which later becomes generalized throughout the body.

Inseparable from this thought is that of the relationship of internal secretions. It is understood that not only the endocrine glands but most every tissue of the body contributes an internal secretion. Accurate knowledge as to whether these hormones influence the tissues of the body so as to be a determining factor in the creation of a state of susceptibility, will be awaited with interest. Until such proof is presented reliance for the present must rest upon a considerable clinical observation. A long list of writers have directed attention to such a relationship, notably John Mackenzie, Solis Cohen, Bates, Pottenger, I. Chandler Walker, Sajous, Zubelin and Grant Selfridge. The beneficial results obtained from the employment of thyroid and pituitary extract, particularly in children, is worthy of attention; also the universal use of adrenalin in asthma and vasomotor rhinitis, suggesting a gland dyscrasia. If sensitization to a given protein cannot be demonstrated, the treatment by gland extract often brings about the desired relief.

Looking at the subject as a nasal reflex there is sufficient anatomic and physiologic information to account for the various vasomotor symptoms. The immense area of peripheral exposure offered by the membranes of the respiratory passages and conjunctival surface favors a degree of irritation and absorption present in no other part of the body. These membranes are supplied with a nerve mechanism of such complexity that the various impressions can be carried to all parts of the body. The nerve fibers that emerge from the sphenomaxillary fossa and that are distributed to the nasal and acces-

sory nasal membranes contain sensory, secretomotor, vaso-
dilator and vasoconstrictor fibers. Brubaker (Jour. A. M. A.,
August 23, 1919) states that the effects of a local disturbance
of the nose may by afferent pathways so enormously stimu-
late the associated central cells as to allow an overflow of im-
pulse into neighboring vasodilator, secretomotor and viscero-
motor centers in the medulla and cord as to cause respiratory,
cutaneous, circulatory and visceral symptoms. Sluder (Jour.
A. M. A., August 23, 1919), quoting Langley, accounts for
the nasal reflex origin of asthma by the impulses from the
nose, passing from the nasal ganglion to the vidian, carotid
plexus and lower cervical ganglion and thence by the way
of the thoracic nerves to the lung. The work of Blos, Yonge,
May, Gordon and my own was principally based on the pre-
vious knowledge that experimental stimulation of Meckel's
ganglion or the peripheral cut end of the great petrosal caused
hyperemia of the nasal membrane with dilatation of vessels
and great increase of secretion. Upon this information has
been built a theory of inhibition, which put into practice
brought about a cessation of the reflex phenomena. In this
is found a satisfactory explanation of the beneficial results
obtained in these cases from the injection of alcohol into the
nerve supply areas of the nose. If the nerves are properly
blocked at the various exits in the nose there will result an
inhibition of the various sensory sympathetic symptoms with
a corresponding cessation of the disorder. The modus oper-
andi of this form of treatment I have presented at various
times.

Now, as to the results obtained by the various methods.
There is no accurate way of obtaining reliable information.
The best that can be done is to rely upon the statement of the
patient. This may be correct and it may be incorrect. From
the use of pollen vaccine treatment, from preparations fur-
nished by commercial houses, the information furnished on
actual results cannot be reliable, because these reports come
from all over the country, from many physicians, used under
varying conditions. In many instances the cases have not
been carefully selected nor properly studied or followed up
and accurate results in some are not known. The observations
of Walker (ANNALS OF OTOLOGY, RHINOLOGY AND LARYN-

GOLOGY, No. 3, 1922), in the preseasonal treatment of 115 grass cases showed about 50 per cent free or practically free from symptoms, and of 420 ragweed pollen cases about 40 per cent free or practically free. The results obtained from treatment during the attack were not as good: 32 per cent practically free in the grass cases, and 14 per cent free or practically free in the ragweed cases. This represents an average result obtained by many others. Besredka and G. M. Mackenzie have shown the value of local desensitization by the application of the antigen to the nasal and respiratory membranes, Parks favoring ragweed pollen for this purpose. Caulfield combines the subcutaneous with the local application.

The bacterial vaccines, especially when used in conjunction with methods tending to eradicate active foci of infection, offer many instances of cures. Even removal of the infectious process without the added vaccine has brought the relief sought for. The work of Schadle, Fink and Helmholz have proven this.

The serum treatment of Dunbar and his followers in this country (Somers, Cohen, Joachim, Loeb, Stein) have shown beneficial results that compare favorably in many instances with those of the vaccine treatment of today.

Mention might be made of the results obtained by Henske with the use of autogenous defibrinated blood. He obtained permanent relief in 56 per cent and temporary relief in 25 per cent of 16 cases of bronchial asthma and hay fever.

With the alcohol treatment injected locally in the nose just prior to the expected attack the beneficial results in 100 cases show 80 per cent free or almost free from symptoms.

This percentage can be improved by more careful selection of cases and a more accurate technic than is always used. It has the advantage of requiring only one or two treatments and therefore, as an economic factor, it has every advantage.

CONCLUSIONS.

Notable progress has been made in the pathogenesis of this disorder.

From the evidence at hand the opinion prevails that the disorder is one of sensitization.

That the antecedent cause of this state of sensitization is as yet not fully understood, but probably there is a particular

altered state of the fluids of the body. Whether this is a chemical change in the elements or simply a disproportion of substances or an excess or lack of some endocrin secretion, the future of physiologic chemistry must determine.

That hay fever symptoms may be due to an anaphylactic reaction or a reflex action.

That many different external causes, acting as the local exciting irritant exist, and in the sensitive create symptoms of hyperesthetic rhinitis. Without both factors actively being present at the same time no symptoms can result.

The use of specific pollen solution, when properly selected, prepared and administered, will materially relieve a small number of hay fever patients. Immunity when obtained is usually temporary, and when so, treatment must be repeated as often as is required. The unskilled administration, as well as the employment of improperly prepared or unstable serologic products may contribute to the sensitization of an individual. Even in the hands of the skilled, alarming and serious results have followed the use of vaccines. The percentage of cures obtained from the employment of pollen vaccines is not greater than results obtained by other methods.

That the results obtained from the nerve blocking method with alcohol give the greatest percentage of freedom of symptoms. That this method of treatment can be given at a much less expense of time and money to the patient. That the possibility of unfavorable results is minimum.

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LXXXII.

BRAIN ABSCESS OF OTITIC ORIGIN IN THE LEFT TEMPORO-OCCIPITAL REGION WITH ALL THE TYPICAL SYMPTOMS. OPERATION. RECOVERY.

By Alexander Zebrowski, M. D.,

New York.

G. S., 50 years of age, complains of severe pains in the left ear and in the left mastoid region, accompanied by fever and otorrhea. All these symptoms appeared suddenly three days ago. However, the otorrhea had persisted for about 20 years but was not constant. The patient says that during the last five years the left ear was always dry. Status praesens: A tall, well nourished man; in the lungs, heart and abdomen nothing abnormal. The right ear, nose and retropharyngeal cavity normal.

The left ear: Voice, 5; whisper, 2. Weber lateralized to the left ear. Rinne negative. Fistula test negative. Profuse purulent discharge.

Otoscopic examination: Drum membrane, red in color and swollen. There is a small round shaped perforation in the front of the mallet. The superioposterior section of the drum is bulging (Fig. 1). Temperature, 103 F.; pulse rate, 116.

On the same day paracentesis of the left drum membrane was made, the incision being through the whole length of the drum.

On the next day the pain in the ear disappeared. Temperature, 100 F. Slight tenderness over the right mastoid region only on pressure. After application of the usual treatment the general condition of the patient improved markedly. The purulent discharge became scanty, the mastoid process painless, temperature normal. Suddenly, ten days after the paracentesis, the patient experienced a terrible headache, confined excusively to the left parietal region. Simultaneously he had nausea and vomiting. Temperature, 102 F.; pulse rate, 72.

The examination of the ear revealed nothing suspicious.

The old perforation was of the same size as previously, a thin scar after the incision being noticed and a scanty mucopurulent discharge is present. The mastoid process was not painful or sensitive, and the hearing on the left side was slightly diminished. Caloric reaction normal. The rise of the temperature and the intense headache lasted for three days. At the same time I noticed some slight changes in the behavior of the patient. Usually rather talkative and disposed to complain of his sufferings, he gradually became taciturn, answered the questions after some hesitation and complained of headache, loss of appetite and sleeplessness. The mastoid process was absolutely not sensitive. Temperature and pulse rate normal. Three and one-half weeks after the purulent discharge appeared, the headache suddenly disappeared and at the same time the patient showed symptoms of amnesic aphasia. Changing from his taciturn disposition, he quite suddenly began to discuss questions which had nothing to do with his illness, using queer expressions and wrong words. The patient stammered, used unrecognizable words and forgot the names of many things, such as "handkerchief," "penknife," "key." It was impossible to state if the alexia and agraphia were present, because the patient constantly refused to write or to read anything. The pulse rate was markedly retarded, 54. Temperature at night, 98 F. The examination of the eye by an ophthalmologist revealed on the left side a marked choked disc, on the right side dilated blood vessels.

Next day a modified radical mastoid operation was performed. After the typical incision, the periosteum was found not thickened and not adherent, the mastoid process totally sclerotic, the antrum of a normal size containing a slight quantity of pus. The roof of the antrum had a normal appearance, no fistula being visible. Careful probing of the tympanic cavity revealed quite smooth surface of the ossicles, no granulations, therefore the ossicles were left in situ. The posteriosuperior part of the external bony meatus was removed up to the attachment of the drum. In the direction of the posterior cranial fossa we met some mastoid cells filled with the pus. The surrounding bone was hard as ivory. The posterior cranial fossa was opened and the wall of the sinus found healthy. The middle cranial fossa was opened, the dura show-

ing a normal appearance. Two punctures anteriorly and two straight were without result. Incision with a scalpel to the depth of 3½ cm. also revealed no pus. The fifth puncture far posteriorly towards the occipital lobe revealed an abscess at the depth of 4 cm. I got in the syringe more than 8 cc. of pus, grayish in color and odorless. The free incision through the dura and the brain tissue evacuated about two tablespoons of pus. Drainage of the abscess cavity by means of iodoform gauze. Panse's plastic. The bacteriologic examination of the pus showed that the infection was produced by the streptococcus pyogenes, pure culture of the streptococcus being found with no mixed infection.

Next day the patient had some headache and the general condition improved. Temperature, 94 F.; the pulse rate, 74 to 68. The third day after the operation dressings were changed. About one tablespoon of a pus, creamy in color and odorless, was evacuated under pressure. The after treatment consisted in the change of the dressing at first every day, afterwards every second day. On the 16th day after the operation the opening in the dura was closed. The after treatment of the wound after the radical operation lasted about three months. The patient, however, returned to his usual occupation four weeks after the operation. The ear was dry.

The symptoms of aphasia and disartria disappeared suddenly on the fourth day after the operation. On this day the patient read the papers and wrote a letter. The retardation of the pulse disappeared immediately after the evacuation of the abscess. On the operating table the pulse rate ran from 54 to 74, and even to 80. The general condition and the appearance of the patient were greatly improved; while in the hospital he gained nine pounds in three weeks. The examination of the eyes showed disc normal. The hearing was fairly good, 5 for the whisper. Behind the auricle a lineal scar was to be seen.

Epicrisis.—The case described is, as I have already stated, a classic example of an otitic brain abscess developed suddenly during a chronic otorrhea. Being under observation from the very beginning of the complication allowed us to state how long it took for the development of the abscess. It is very likely that this time was at any rate shorter than four weeks,

because the first symptoms appeared earlier than a month
after the previously dry ear began to discharge. Such a short
space of time has already been described, by Jansen in his
textbook, a case of brain abscess, the patient being 69 years
old, in which a rupture into the lateral ventricle occurred 14
days after the previously healthy ear began to discharge.

The disturbances of speech, disartria and aphasia appeared
very early and were markedly developed. The operation re-
vealed that the abscess was located far away from the center
of speech—it was found in the posterior part of the left tem-
poral lobe in the occipital region. The aphasia was probably
due to the collateral edema and encephalitis. After the evac-
uation of the pus the speech of the patient was normal on
the fourth day. In this case the disturbances of speech—
usually the most important symptom for the localization of a
left sided brain abscess—did not assist in locating the abscess.
It was found only after four unsuccessful punctures and not
in the place where presumably it should be found. In the
cases of otitic left sided brain abscess which I have previously
described (Monatschrift f. Ohrenheilkunde, 1905 and 1911),
the abscess was always found very near to the center of
speech.

In this case the infection of the brain was due to the strep-
tococcus pyogenes. In the article of Blau*, a comparative
study of 1,000 cases of endocranial complications of chronic
purulent otitis media, in 218 cases where a bacteriologic exam-
ination was made, in 68 the infection was due to the strepto-
coccus pyogenes. In my case it was practically a pure culture
of these bacteria, no mixed infection being present. This in-
dicates that the abscess was recent.

The otoscopic picture in my case was quite unusual, because
in the monograph of Blau cited above we do not find an
analogous case. According to Blau, the endocranial compli-
cations are nearly always accompanied by the large or total
destruction of the drum.

Among the 1,000 cases of Blau there were 234 brain ab-
scesses. The eyes were examined in 135 cases—the choked

*Louis Blau: Zur Lehre von otogenen intrakraniellen Erkran-
kungen.   Beitraege zur Anatomie, Physiologie, Pathologie und
Therapie des Ohres, der Nase und des Halses, 1917, page 86.

disc was found only in 14 per cent of the cases. In 46 per
cent of the cases the disc was normal, in 40 per cent the
changes were but slight, dilated blood vessels or optic neuritis.

It is interesting to trace the way through which the infec-
tion reached the posterior cranial fossa. In this case the
infection of the brain followed the disintegration of some
pneumatic cells located beyond the limits of the mastoid
process, near to the occipital bone. Blau (l. c.) in 1,000 cases
of intracranial complications cites only one case of brain ab-
scess in which the pus infected the middle cranial fossa by
disintegration of pneumatic cells in the same region. Usually,
as we know, the pus enters the middle cranial fossa through
the tegmen of antrum or tympanum, according to Blau, in
134 cases among 234 abscesses of the brain (l. c. page 118).

FIGURE 1.

1. Old perforation.
2. Bulging of the drum sack.
3. Incision.

LXXXIII.

SOME GENERAL EFFECTS OF LOCAL ANESTHET-
ICS ADMINISTERED AS IN TONSILLECTOMY.

By Ellison L. Ross, Ph. D., M. D.,

Chicago.

Symptoms of the systemic effect of local anesthetics fre-
quently appear during the removal of tonsils. Sometimes this
effect is hardly noticeable and at others it is very pronounced.
In the mildest cases there is a tendency to tremble and a feel-
ing of weakness after the operation. In the more severe cases
this is intensified, definite systemic effects are noticeable;
faintness appears, the face turns pale and may show some
cyanosis, breathing is rapid and the pulse is scarcely percepti-
ble to the touch. The occasional severity of these symptoms of
intoxication has aroused interest, and the author determined
to ascertain as far as possible the primary cause and the
mechanism of these reactions.

The general toxic effect of cocain and the occurrence of a
high degree of susceptibility to the drug of some individuals
are of common knowledge. This has led to the exercise of
great care in its use. Today few inject cocain for any pur-
pose. Its use is confined chiefly to surface applications in
limited amounts. It is common practice to paint the pillars
of the tonsils with a 10 to 20 per cent cocain solution before
the local anesthetic is injected for a tonsillectomy. The local
anesthetic usually injected is procain (novocain) with some
adrenalin. So intoxication may arise from one, two or three
drugs acting individually or synergistically.

The problem is emphasized by Dr. T. J. Harris[1] in the re-
port of a fatality. Cocain, gr. 1/12, and adrenalin, 8 to 10
minims, were injected. Death occurred quickly. The heart
seemed to stop a considerable time before the respiration.
The autopsy showed enlarged glands in the axilla and groins
and the thymus weighed 18 grams. Dr. Harris reported the
cause of death as the result of "overdilated right auricle due

to an enlarged thymus gland, with cocain and adrenalin as an
exciting cause." In the discussion Dr. Hubbard reported a
death from 15 to 20 minims of adrenalin and thought that
Dr. Harris' case resulted fatally from the adrenalin. Dr.
Swain stated that he had treated a case of enlarged thymus
with adrenalin and could see no "incompatibility between the
two." He was inclined to think the cause of death of Dr.
Harris' patient was the adrenalin. Dr. Roe, in discussing
the case, thought that the death was due to hypersensitiveness
to cocain. Dr. Harris, in closing, stated that he thought that
the cocain had no deleterious effect in his case. This discus-
sion by experienced men of the action of commonly used
drugs which resulted in death emphasizes the incompleteness
of our knowledge of the clinical action of these drugs and
the importance of the subject.

Everyone recognizes the toxic effect of cocain and its varia-
bility in different individuals. Adrenalin, alone, is used rather
freely with little thought of any toxic action. However, Bux-
ton[2] in his book on anesthetics says that even minute doses
of adrenalin are said to cause unpleasant effects—e. g., giddi-
ness, fainting and collapse. Mortimer[3] discussed the dangers
from adrenalin as viewed by the pharmacologist and the
harmlessness of the drug as viewed by the physician. He
concludes that the question is an unsettled one, and the safest
procedure is not to inject adrenalin but use it for surface appli-
cations only. Allen[4] in his book on anesthesia states that
adrenalin may prove a distinct advantage after cocain. He
quotes J. M. Berry as agreeing with him. He mentions Thriss,
Niles and Muhlberg and Petrow as considering the action of
adrenalin to be that of a beneficial stimulant after cocain. As
far as we are able to learn, no clinician has suggested synergy.

Pharmacologists agree that cocain and adrenalin act syner-
gistically on blood pressure. If adrenalin and cocain are in-
jected into a vein the rise in blood pressure is more than the
sum of the increases produced by the adrenalin and cocain if
injected separately and a considerable time apart. Cocain seems
to sensitize the sympathetic nervous system so that the stimu-
lation by adrenalin produces a much greater reaction. These
facts led us to suspect considerable rise in blood pressure due
to drug action during the process of a tonsillectomy under

local anesthesia.   From the clinical side, there seems to be
some doubt on this point.   It is usually hard to feel the pulse
when a toxic disturbance results from injections of a local
anesthetic, and the common conclusion is that the blood press-
ure is low.   Naturally, with fainting and respiratory disturb-
ance, a low blood pressure is expected, since these are marked
symptoms always present with severe hemorrhage.   Usually
with moderate increases in blood pressure there is an increase
in buoyancy and a lack of any disturbance of respiration.

Recently the author made a series of experiments[5] on dogs
to determine the effect of the local anesthetic when adminis-
tered in the usual clinical manner.   In Test I a number of
chloretoned animals were injected in the tonsillar region with
a dose of adrenalin.   As soon as the effect of this drug wore
away a dose of adrenalin with cocain was injected.   Observa-
tions were made on the arterial and intracranial venous blood
pressures, heart action and respiratory rate.   The average re-
sults are represented on the chart.   Test II was made on an-
other group of dogs, in which moderate doses of morphin and
atropin were given half an hour before the same treatment as
the first group.   The average results are represented parallel
with those from Test I.   Test III was made on a third group
of dogs.   This group was given the usual dose of adrenalin
in the usual way, used in Test I, and after the effect of this
had worn off cocain was injected.   The results are represented
parallel with those of the preceding tests.   The values are ex-
pressed in per cents of the normal.

Tests I, II and III determine the effect on arterial and intra-
cranial venous blood pressures, heart action and respiratory
rate, of injections in the tonsillar region of adrenalin, cocain,
adrenalin and cocain, and adrenalin and cocain preceded by
morphin and atropin.   The arterial pressure was increased 8
per cent by adrenalin alone, 83 per cent by cocain, 223 per cent
by adrenalin and cocain, and 202 per cent by adrenalin and
cocain preceded by morphin and atropin.   From this the syner-
gistic action of adrenalin and cocain will be calculated as 132
per cent.   The injection of morphin and atropin affected the
combined action of adrenalin and cocain relatively very little.
It is striking that the arterial pressure is so enormously
increased.

The intracranial venous pressure was unaffected by adrenalin, but increased 100 per cent by cocain, 467 per cent by cocain and adrenalin, and 282 per cent by adrenalin and cocain preceded by morphin and atropin. This enormous increase in venous pressure due to adrenalin and cocain is more than twice as great as that taking place in the arterial system. This condition of affairs is very significant. Whenever the intracranial venous pressure increases twice as much as the arterial pressure, there is sure to be intracranial circulatory stasis and a resulting asphyxia of the central nervous system. Asphyxia of the respiratory center would increase respiration and give a feeling of smothering to the individual. Our tests 'show that the rate of respiration was increased 100 per cent by adrenalin and cocain. A feeling of smothering is a common complaint where there is any considerable reaction from the use of cocain. The failure of adrenalin and cocain to produce any increase in respiration when following morphin and atropin is due to the action of morphin to reduce the sensitiveness of the respiratory center. Asphyxia of the higher parts of the brain would lead first to a feeling of uneasiness and some degree of excitement, which would quickly be replaced by a feeling of faintness and dimness of vision. This is the common experience of the individual who suffers a reaction from the use of adrenalin and cocain. The changes in the heart action caused by these injections are of little importance. The slight slowing of the heart action with an increased amplitude of beat seems to be only compensatory changes.

The above tests were made by injecting adrenalin and cocain. On account of deaths resulting from the injection of cocain, it is chiefly used in the practice of medicine only on exposed surfaces. The question as to whether the local application of cocain in the usual manner about the tonsils would exert any influence on later injections was yet to be answered. Test IV was made on a fresh group of dogs. Adrenalin was injected into these dogs, and after its action had worn away, the throat was swabbed with 20 per cent cocain. Shortly after the cocain a second injection of adrenalin was made. Test VI was made on another group of animals, which was given adrenalin followed by swabbing with cocain. This test enabled us to measure the action of cocain alone. Tests IV and VI

furnish data by which we might measure the influence of throat swabbing with cocain on the action of injected adrenalin.

The arterial pressure was unaffected by the dose of adrenalin used on this group of dogs when no cocain had been applied. Swabbing with cocain after the action of the first dose of adrenalin had worn away produced an increase in arterial pressure of 16 per cent, and the repetition of the dose of adrenalin closely following the cocain produced an increase of 137 per cent. The synergistic effect of the cocain when swabbed on and the adrenalin injected was very great on the arterial pressure.

The changes in intracranial venous pressure produced in Tests IV and VI are similar to those of the arterial pressure. The first injection of adrenalin produced a very slight drop (7 per cent), and the second injection produced an increase of 193 per cent. The rise in intracranial venous pressure was very much greater than the rise in arterial pressure. The same condition is brought about by swabbing with cocain as by injecting with the cocain.

The question that naturally arises is whether swabbing of the throat with cocain, novocain and adrenalin would produce an important rise in blood pressure. Test V was made on a new group of dogs. These dogs were injected with adrenalin and novocain. After the action of these drugs had subsided cocain was swabbed on the throat, and following this the first injection was repeated. This test gave approximately the same results as without the use of novocain, namely, a rise in blood pressure.

The results of these six tests indicate clearly that cocain swabbed on the throat preceding an injection containing adrenalin has a very powerful influence on arterial and intracranial venous pressure. The intracranial venous pressure is much more increased than the arterial pressure. This we consider as the possible primary cause and mechanism of temporary disturbances after the use of these drugs.

The fact that all of the preceding tests were made on dogs and not men, and the fact that all of the dogs were under general anesthesia, make it possible that the conclusions reached from the described experiments are not applicable to the con-

ditions met with in the removal of tonsils under local anesthesia in the usual manner. To test this possible objection, blood pressure measurements were made on a number of patients during tonsillectomy.

Systolic blood pressure measurements were made at intervals during tonsillectomies, done by four different operators. The first measurement was made in the operating room before the operation. The second measurement was made just before the dissection of the first tonsil. The third measurement was taken just before the dissection of the second tonsil and the last measurement occured at the end of the operation.

The drugs used by all operators were the same. Cocain and adrenalin were swabbed on the throat before the injection of novocain and adrenalin. The amount and method of administration of the drugs were different with the operators. Operator A swabbed the throat with 8 per cent cocain and a small amount of adrenalin. He injected ½ per cent novocain containing about 5 per cent of adrenalin (1:1,000). He always injected both sides before starting the operation. All of his cases received ⅛ grain of morphin and 1/150 grain of atropin three-quarters of an hour before the operation. Operator B swabbed the throat with 10 per cent cocain with 50 per cent adrenalin (1:1,000) before injecting novocain—1 per cent, containing a little over 1 per cent adrenalin (1:1,000). One side was injected and operated on before the second was injected. Operators C and D swabbed with 20 per cent cocain and a small amount of adrenalin before 2 per cent novocain and about 4 per cent adrenalin (1:1,000) were injected. In some cases both sides were injected before any operating was done, and in others one side was injected and the tonsil removed before the other was injected. In Table I is recorded the individual initial, maximal and final blood pressures taken.

Referring to Table I, it is clear that there was always a rise of blood pressure. The amount of rise in blood pressure was quite variable, from 20 mm. of mercury to 114. In 18 cases out of the 23, the maximal pressure occurred before the removal of the second tonsil.

Tables II and III contain the average measurements. In Table II the results are expressed in millimeters of mercury, and in Table III they are expressed in percents of the normal.

The average blood presure for all 23 cases were 119.8 mm. before operation, 160.8 mm. at the height of rise and 140.8 mm. at the close of the operation. The average maximum of rise was 34.2 per cent and the average increase in blood pressure at the end of the operation wac 17.6 per cent. This maximum of increase is considerable and should be considered as a possible source of danger in some cases. Certainly, these measurements indicate that efforts to prevent mishap from the use of local anesthetics should not be directed toward increasing the blood pressure but toward preventing a rise in it.

There were three cases in this series that had rather severe fainting reactions. The average blood pressure of these before operation was 128 and the average pressure raised to 194 mm., and at the end of the operation the average blood pressure was 162 mm. of mercury. The maximum of pressure was an increase of 51.5 per cent, and the final pressure was 26.5 per cent above the initial pressure. These few cases certainly substantiate the view arrived at by animal experimentation that the rise in blood pressure is the cause of the temporary disturbances produced by local anesthetics.

Some operators consider that there will be less reaction from the local anesthetics in tonsillectomies if one side is injected and operated on before the other side is injected. Of the series of cases, 13 were so managed and 10 received injections on both sides before dissection was begun. The first group began with a blood pressure of 115.9 mm., which raised to a maximum of 156.8 mm. and ended with a pressure of 131.8 mm. The second group began with a pressure of 124.9 mm., which raised to 166 mm. and then finished with a pressure of 152.4 mm. The maximum of rise in the first group was 35.3 per cent, as compared with 32.9 per cent in the second group. The final pressure in the first group was 13.7 per cent above normal, while that of the second group was 22 per cent above normal. Such results do not argue very strongly in favor of injecting one side and operating on it before injecting the second side.

There were some variations in the work of the several operators. Operator A used more adrenalin and less novocain than the other three, and he always injected both sides at once. Operators C and D used more adrenalin and novocain than

Operator B. The maximum of increase for Operator A was 52.5 per cent, for Operators C and D 36.5 per cent, and for Operator B 26.5 per cent. These values grade in the same general ratio as the amount of adrenalin used. Other variations in the performance of the operators and changes of blood pressure did not seem to be of significance.

SUMMARY AND CONCLUSIONS.

The administration to animals of local anesthetics with the usual clinical technic practiced in the removal of tonsils, produces marked circulatory changes. The arterial pressure is greatly increased, as would naturally be expected from the well known synergistic action of cocain and adrenalin when injected into the blood stream. The intracranial venous pressure is relatively very much more increased than the arterial pressure. The arterial pressure was increased 223 per cent, while the venous pressure was increased 467 per cent. If either cocain or adrenalin be omitted the circulatory changes are negligible. In the previous paper[5] it was pointed out that the great variations in blood pressure were capable of producing marked mental and respiratory disturbances.

Measurements of blood pressure in people undergoing tonsillectomies in which cocain, adrenalin and novocain were used indicate a regular rise or blood pressure of considerable amount but subject to wide individual variation. Those cases which experienced considerable reaction showed much greater increase in blood pressure.

These observations lead us to conclude that the minimum of cocain, to be of any value as an anesthetic, is sufficient to greatly sensitize the body to the action of adrenalin on blood pressure. Therefore cocain should be eliminated completely wherever possible. When cocain must be used to reduce reflex action of gagging, it may be applied some time after the injection of the adrenalin and novocain, when no synergistic action will result. The amount of adrenalin with the novocain should be reduced to a minimum. The strength of the novocain injected up to 2 per cent seems to be of small significance.

TABLE I.

INDIVIDUAL BLOOD PRESSURE VARIATION DURING TONSILLECTOMY USING LOCAL ANESTHESIA.

No. Patient	B. P. Before Op.	Maximum B. P. Amt.	Time	B. P. After Op.	
1 C-D	137	200	Before 2nd	100	Faintness.
2 C-D	120	234	Before 2nd	168	
3 B	130	150	Before 2nd	147	
4 B	115	160	Before 2nd	130	
5 A	150	194	Before 2nd	180	Both sides injected before operation. Very faint.
6 C-D	114	154	Before 2nd	138	Faintness.
7 C-D	122	158	Before 2nd	116	
8 A	190	238	After 2nd	238	Both sides injected before operation. Morphin-atropin before operation.
9 C-D	105	130	After 2nd	130	
10 C-D	115	135	Before 2nd	125	
11 A	120	165	Before 2nd	160	Both sides injected before operation. Morphin-atropin before operation.
12 C-D	116	172	Before 2nd	162	
13 C-D	100	120	Before 2nd	120	
14 C-D	90	125	Before 2nd	120	Both tonsils injected before operation.
15 C-D	130	160	Before 1st	105	Both tonsils injected before operation.
16 C-D	110	135	Before 2nd	118	
17 A	135	190	Before 2nd	173	Both tonsils injected before operation. Morphin-atropin before operation.
18 A	126	150	Before 2nd	145	Both sides injected before operation.
19 C-D	122	150	After Op.	150	
20 C-D	100	140	Before 1st	110	
21 A	118	170	Before 2nd	160	Both sides injected before operation. Morphin-atropin
22 C-D	90	128	Before 2nd	118	Both tonsils injected before operation.
23 C-D	100	140	Before 2nd	125	Both tonsils injected before operation.
Average	119.8	160.8		140.8	

TABLE II.

AVERAGE BLOOD PRESSURE VARIATION DURING TONSIL-
LECTOMY USING LOCAL ANESTHESIA.

	B. P. Before Op.	Maximum of B. P. Amt.	Maximum of B. P. Increase	B. P. After Op.	No. of Cases
All Cases	119.8	160.8	41.0	140.8	23
Fainting Cases	128.0	194.0	66.0	162.0	3
One Side Injection	115.9	156.8	40.9	131.8	13
Both Sides Injection	124.9	166.0	41.1	152.4	10
Operator A	121.0	184.5	63.5	176.0	6
Operator B	122.5	155.0	32.5	138.5	2
Operators C and B	111.4	152.1	40.7	127.0	15

TABLE III.

AVERAGE BLOOD PRESSURE VARIATION DURING TONSIL-
LECTOMY USING LOCAL ANESTHESIA.

Results expressed in percent of normal.

	B. P. Before Op.	Maximum of B. P. Amt.	Maximum of B. P. Increase	B. P. After Op.	No. of Cases
All Cases	100	134.2	34.2	117.5	23
Fainting Cases	100	151.5	51.5	126.5	3
One Side Injection	100	135.3	35.3	113.7	13
Both Sides Injection	100	132.9	32.9	122.0	10
Operator A	100	152.5	52.5	145.4	6
Operator B	100	126.5	26.5	113.0	2
Operators C and D	100	136.5	36.5	114.0	15

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Percent

Cocain - - - -
Adrenalin ————
   "   after Cocain —— ——
   "   and   "   ———
   "   "   "   after Morph-Atrop —··—
   "   "   Novocain. ++++++++
   "   "   "   after Cocain ——·——

500

400

300

Tests 1,2 and 3.

200

100

200

Tests 4 and 5.

100

200

Tests 5 and 6.

100

Arterial Pressure.   Venous Pressure.   Heart Rate.   Heart Amplitude.   Respiration Rate.

Animal Experiments.
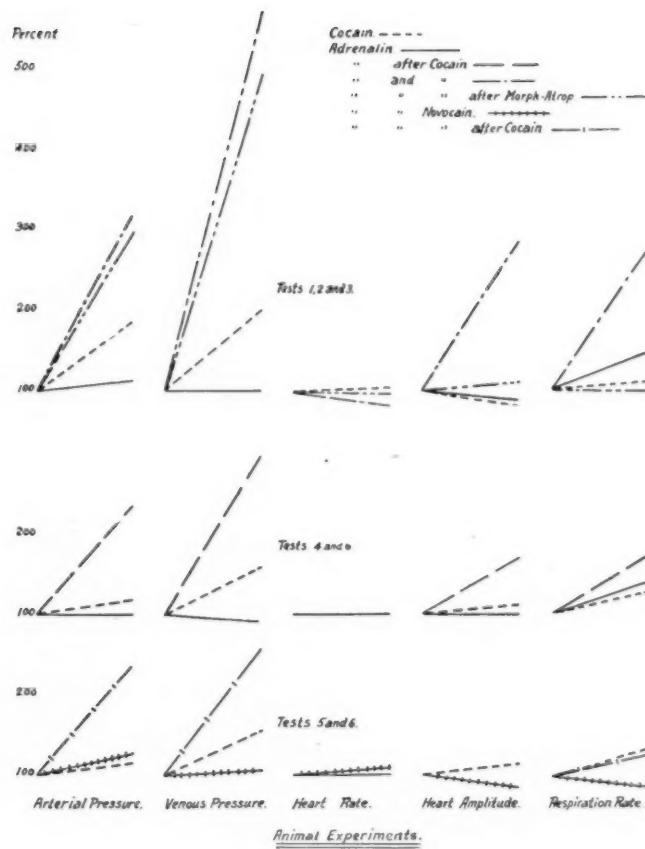
LXXXIV.

TUMOR OF THE CAROTID GLAND WITH STOKES-ADAMS SYNDROME.

By G. W. Boot, M. D.,

Chicago.

The members of this society will remember the patient I demonstrated before them in 1921 as a case of tumor of the neck with Stokes-Adams syndrome. He was a mulatto, aged 42, who was admitted to Cook County Hospital complaining of pain in the left side of the neck and cramps of the muscles on the same side of the neck. His symptoms had been present for the preceding six months. The onset was sudden, with cramps in the muscles of the neck and unconsciousness lasting about a minute. Breathing was very difficult during the attack and the neck was painful. Since that time he had from one to three attacks daily. At times he would have intervals of several days during which he was free from attacks. The pain in the neck and discomfort in breathing were relieved when the neck was flexed. At times the pain extended over the entire left side of the face.

He had acute suppurative otitis media in 1901, malaria in 1908 and gonorrhea in 1911. He had been married sixteen years and had a wife and two children, living and well. His hearing was impaired in the right ear. He vomited occasionally. He had lost 23 pounds during the past six months. He had sexual weakness to the extent that he had had no erections during the preceding six months. He had had no cough before the onset of these spells. He had had no pain in the chest and no night sweats.

Examination showed a small tumor about 2 cm. in diameter in the left side of the neck about its middle and anterior to the sternomastoid muscle. This tumor was painful, and when touched the patient coughed and became weak. During the examination the patient became unconscious for about 30 seconds and frothed at the mouth but had no general convulsion. There was paralysis of the left vocal cord.

Diagnosis by the intern, extrinsic tumor of the larynx with epilepsy.

Examination by the resident confirmed the above findings. His diagnosis was extrinsic tumor of the larynx, left abductor paralysis, with possible epilepsy or hysteria.

Examination by myself was difficult for the reason that any attempt to use the throat mirror brought on a convulsive action of the muscles of the neck and larynx with difficult respiration, and it was only after several attempts that I was able to see that there was no movement of the left vocal cord outward. During this examination I discovered that pressure on the tumor would bring on a convulsive attack during which his pulse became slow. On listening to his heart with a stethoscope, the sounds became faint, and if the pressure on the tumor was long enough continued the pulse could not be felt at the wrist, the heart sounds stopped and the patient had a general convulsion. The condition was that of Stokes-Adams syndrome from vagus inhibition.

The tumor was operated on under the impression that I was dealing with an enlarged lymphatic gland that was in intimate connection with the vagus so that there was partial paralysis of the left recurrent nerve from pressure.

The operation proved to be much more difficult than was anticipated. The tumor was found to lie in the bifurcation of the carotid and intimately to surround the common, internal and external carotids and internal jugular vein so that it could not be separated from them. It was necessary to ligate the common carotid, the internal and external carotid and the internal jugular vein before the tumor could be removed.

I do not know whether the vagus was divided or not, but it probably was severed. Trouble was anticipated from this source but apparently nothing of consequence occurred. His symptoms remained much the same after the removal of the tumor except that he had no further attacks of Stokes-Adams syndrome, and his pulse was more rapid. He coughed a good deal and died about a week after the operation.

At the postmortem it was found that he had acute edema of the epiglottis, glottis, vocal cords and larynx generally; large ulcerative, purulent tubercular cavity of the upper lobe of the right lung; multiple abscesses of the lower lobe of the

left lung; edema and hyperemia of both lungs; acute purulent tracheobronchitis; apical fibrous pleurisy on the left side; right hydrothorax; fibrous perisplenitis; cirrhosis of the liver and fibrosis of the spleen.

The tumor removed was about 3 cm. long, 2 cm. wide and 1.5 cm. thick. It could not be separated from the carotids. The microscopic diagnosis of the tumor removed was in doubt. The pathologists finally concluded to call it carcinoma of the carotid gland.

The carotid gland is a small body about the size of a navy bean, lying at the bifurcation of the common into the internal and external carotid arteries. Its beginning has been seen in the 4 cm. fetus. It is one of the chromaffin bodies, so-called, because of the affinity of its cells for the salts of chromium used in fixing. On this account sections of the carotid gland stain better when fixed in Mueller's fluid than when fixed in formalin. It is composed of cells with large protoplasmic bodies grouped into clusters or strands of various sizes, and lying in alveoli of connective tissue. Its cells somewhat resemble epithelium. It has many capillaries with thin walls. The carotid gland is connected to the carotid artery at its bifurcation by a sort of pedicle composed of fibrous and vascular structures. Its blood supply is by three or four small arteries that enter at its lower pole. A corresponding number of veins leave at its upper pole. Its nerves are numerous and come from several sources, among which are the vagus, the sympathetic, the hypoglossal and glossopharyngeal. Like other chromaffin organs, it possesses a few ganglion cells. The function of the carotid gland is unknown, but the paraganglions have to do with controlling blood pressure. The paraganglions include the tympanic gland, the carotid gland, Wiesel's cardiac paraganglion, the aortic paraganglion or Zuckerkandl's organ, and Luschka's coccygeal gland.

Tumors of the carotid gland are uncommon. The first tumor was reported by Marchand in 1891. Keen and Funke in 1906 collected 29 cases. Callison and Mackenty in 1914 brought this number up to 60. Reid in 1920 found reports of 66 cases and added 3 more from the clinic of Prof. Halsted of Johns Hopkins.

Tumors of the carotid gland may be benign or malignant. The benign tumors are simple hyperplasia, adenoma and angioma. The malignant tumors have been variously diagnosed as perithelioma, endothelioma, epithelioma, perithelial angiosarcoma, sarcoma, carcinoma, and so on.

These tumors vary in size from an almond to a goose egg. They are usually smooth and oval in outline, but sometimes are lobulated when they have been called potato tumors and are probably sarcomas or carcinomas. They are usually dark red in color on section but may be almost white or yellowish. Only one case is on record where they were bilateral. They occur in about equal frequency in the two sexes and on the two sides of the neck.

The symptoms caused by them are deformity; pressure symptoms, such as bruit and thrill, tinnitus aurium, hoarseness, cough and vocal cord paralysis from involvement of the recurrent nerve, contracted pupils from involvement of the sympathetic, dysphagia, and dyspnea.

In the diagnosis the following points are to be considered: The location at the bifurcation of the common carotid; movability laterally but not up and down; ovoid shape; usually smooth; usually single; transmitted pulsation; bruit and thrill; bulging of the lateral wall of the pharynx; pupils usually constricted; slow growth (cases have been reported lasting 8, 9, 16 and 40 years); absence of tenderness and pain, as a rule; slowing of the heart—i. e., Stokes-Adams syndrome; hoarseness and cough from vocal paralysis; pigmentation of the body in some cases.

Differential diagnosis should include consideration of the possibility of enlarged lymph glands—i. e., cervical adenitis, gland metastases in carcinoma, Hodgkins disease, branchial cyst, syphilitic enlargement of glands, gummata, tubercular glands, dermoids, aneurism. Many of these cases give the history of a lump in the neck for many years, with very slow growth. Finally the lump takes on rapid growth.

If these tumors are to be treated successfully they should be operated on early before they have become so intimately attached to the carotids that ligation of the carotids is necessary for their removal. The surgeon will rarely see them in this stage and will still more rarely make the correct diag-

nosis. Ligation of the common carotid is a serious operation though not necessarily fatal. I have ligated the common carotid twice, once for pulsating exophthalmus and once for tumor of the carotid gland. In the former case the patient recovered; in the latter, death was not the result of ligation but of the concomitant pulmonary condition.

In my case I feared the result of cutting the vagus, but my fears were not well founded. Before operating, my patient was given a large dose of atropin with the idea of suppressing the inhibition of the vagus. Possibly this was the reason why no serious trouble was encountered with this nerve.

The results in the cases operated on up to date have not been very creditable to the surgeon. The mortality is high, and laryngeal and other disturbances of a permanent nature should make one hesitate before operating. Among the serious complications that have resulted are hemiplegia, aphasia, paralysis of the facial nerve and of the hypoglossal nerve. These tumors recur rather frequently. Most cases have been operated on under mistaken diagnosis. In my case the operation was done in the attempt to relieve the patient of distressing epileptiform choking spells from which he suffered and which completely incapacitated him from work.

25 E. WASHINGTON ST.

LXXXV.

KLEBS-LOEFFLER INFECTION OF THE MIDDLE EAR AND MASTOID.

By Russell Webber, M. D.,

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An examination of the literature shows but few recorded cases of true primary diphtheritic otitis. The textbooks mention cases of Klebs-Loeffler infection of the middle ear following cases of pharyngeal diphtheria, but practically no reference is made to Klebs-Loeffler infection not dependent on a known primary pharyngeal or nasal infection.

The first mention of the possibility of diphtheria of the middle ear unassociated with pharyngeal diphtheria is found in Billington's book published in 1889. Ballenger refers to cases of diphtheritic otitis sequelæ of pharyngeal diphtheria, but does not mention an infection primary in the ear. Phillips also dwells at some length on second diphtheritic infection and briefly dismisses primary infection in the ear by observing that it happens very rarely. Kerrison goes into the secondary otitis of diphtheria in some detail but does not refer to a primary infection.

In the usual form, an infection secondary to pharyngeal diphtheria, the otitis appears during the course of the disease. Epidemics of diphtheria vary greatly in the proportion of ear complications observed. Kerrison says that about 5 per cent of diphtheria cases develop some form of ear lesion. These lesions are much less extensive than those following measles and scarlet fever, and while there may be extensive destruction of the membrana tympani itself, mastoiditis is less apt to occur.

J. J. Thompson in 1914 reported eleven cases of diphtheritic or pseudodiphtheritic mastoid wound infection, all occurring during a period of six months. None of the cases had pharyngeal diphtheria. In but one of these cases did people coming

in contact with it develop diphtheria, guinea pigs injected with cultures from the wounds did not die, and no change occurred in any of the cases in which diphtheria antitoxin was used. Of all these cases, but two seem to be described as being positively Klebs-Loeffler infection. Thompson's finding that these cases were unaffected by the injection of antitoxin differs from the experience of other observers.

Pugnat describes primary diphtheritic otitis media and mastoiditis occurring as a sequel. All of the cases that he has observed cleared up rapidly with the injection of diphtheria antitoxin.

W. C. Bane of Denver, Colo., reports what he believes to be one of the few cases of true diphtheria of the ear. The course of the case was that of the usual acute otitis media. The patient had had pain in the right ear followed by rupture of the membrana tympani and drainage before being seen. Cultures taken from the aural discharge showed the presence of diphtheria bacilli and antitoxin was given. A general subsidence of the symptoms followed, but the bacilli persisted in the discharge for two months, the left ear finally becoming involved. This is one of the cases in which the injection of diphtheria antitoxin did not cause the disappearance of Klebs-Loeffler organisms from the discharge.

Blanchard reports a case of double diphtheritic otitis media. For eight days the case, apparently one of ordinary acute otitis media, did not respond to the usual treatment. He ran a high temperature and got progressively weaker. On the ninth day a peculiar exudate was noted in the ears, and when cultures were taken a Klebs-Loeffler infection was discovered. Antitoxin was administered and not only did the general symptoms subside very rapidly, but the discharge ceased in both ears within four days.

In Alfred Kahn's case there was a diphtheritic membrane surrounding and extending up and through the eustachian tube of the right ear. The process continued into the middle ear with destruction of the membrana tympani. An X-ray picture taken at the time showed extensive mastoid involvement, but operation was delayed until the effect of diphtheria antitoxin could be noted. Such marked improvement resulted

that operation was not resorted to, and the condition eventually cleared up completely.

Dr. Vinsonhaler of Little Rock, Ark., reports two cases of Klebs-Loeffler infection of the middle ear, one case eventually becoming diphtheritic mastoiditis. In neither case was there any pharyngeal diphtheria. In both cases all the severe general symptoms subsided with the injection of antitoxin, and within a few days diphtheria bacilli were absent from the discharge.

Two cases coming under my own observation are of interest.

Case 1.—C. M., aged 52. First seen February 5, 1920. Twenty years previous to this time patient had had an incomplete mastoid operation performed on the right side, and up till present illness had had no further trouble from this ear. About the middle of January, 1920, he had an attack of facial erysipelas, which had almost cleared up when seen. A few days before I was called to see him, his right ear began to discharge but he suffered no particular pain. On February 4th his temperature rose to 102, and he complained of severe discomfort in his right ear. When I examined the ear I found the canal filled with a thin watery discharge, but did not see any sign of anything resembling a diphtheritic membrane. There was no mastoid pain or tenderness. Examination of nose and throat was negative. I ordered frequent hot irrigations of boric solution and took a culture from the ear. When the laboratory report returned "Klebs-Loeffler," I felt sure a mistake had been made and my culture mixed with another, but a second culture gave the same result. Ten thousand units of antitoxin were given, and within thirty-six hours the patient's temperature dropped to normal and the discharge ceased. Two weeks later discharge recommenced. This time cultures were taken from the ear, nose and pharynx. All were positive for Klebs-Loeffler, though no membrane could be found in any of these sites. After a second dose of antitoxin the temperature returned to normal and the discharge diminished. A week later definite mastoiditis developed and the right mastoid was operated upon. At no time, then or thereafter, did the bacteriologist report presence of diphtheria bacilli, though frequent cultures were taken.

Case 2.—F. A., aged 3. Child was admitted to the Waterbury Hospital January 24, 1922, with discharge from both ears, pain and swelling in both mastoid regions, and a temperature of 104.4°. A double Schwartze was done by my colleague, Dr. Munger, immediately after patient was admitted. Both mastoids were very congested, granulation tissue was found in both antra, but there was no great outpouring of pus. Cultures taken from the mastoid antra were reported to be staphylococcus aureus. For days following operation there was no marked change in the child's condition. Temperature varied from 100 to 102 degrees. His movements were sluggish and his appearance was that of a very ill child. There were no symptoms to suggest intracranial complications. Eye grounds were clear and urine was practically normal. A blood culture was taken and staphylococcus aureus reported. Blood count was normal, aside from a reduction of the red blood cells, which were down to 3,000,000. It was decided to give the child an autogenous vaccine, made from the blood culture. The first dose was given on February 6th and there was a very marked reaction. Both mastoid wounds were again cultured and Klebs-Loeffler was reported in both but was absent from nose and throat. As the patient was already getting the vaccine for the blood infection and as he had had very severe reaction to it, but one dose of antitoxin was given and that but 5,000 units. Thereafter, the temperature rapidly subsided, although diphtheria bacilli persisted in the mastoid wounds until the 20th of March, when they were finally reported free. Child made an uneventful recovery.

The following conclusions may be drawn from a study of these cases:

1. That although the small number of recorded cases testifies to the rarity of the condition, diphtheria of the middle ear may occur without known previous Klebs-Loeffler infection elsewhere.

2. That a more careful bacteriologic examination of aural discharges in obscure cases may reveal the presence of diphtheritic infection otherwise overlooked.

3. That practically all cases of aural diphtheria respond in a marked manner to the injection of diphtheria antitoxin. In

not all cases does the Klebs-Loeffler organism disappear from the discharge, but in the cases reported the general symptoms did subside when antitoxin was administered. Hence in all cases where there is any question as to whether the infection is true Klebs-Loeffler or pseudodiphtheritic, antitoxin should be given.

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SOCIETY PROCEEDINGS.

THE CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

Meeting of Monday, April 2, 1923.

THE PRESIDENT, DR. CHAS. H. LONG, IN THE CHAIR.

Nasal Obstruction Which Apparently Had Affected His Mental State.

DR. C. H. LONG presented a patient who had been operated on for nasal obstruction, which apparently had affected his mental state. The patient insisted that there was a cartilage which obstructed his left naris and that he was unable to get free passage of air through his nose.

Foreign Body in the Esophagus.

DR. JOHN A. CAVANAUGH reported a case of foreign body in the esophagus in which a diagnosis of carcinoma of the esophagus had been made, and presented skiagraphs showing the hourglass appearance and obstruction of the esophagus. The history was that the patient awoke one morning and had difficulty in swallowing with regurgitation of food. He had no knowledge of having swallowed a foreign body, but had been on a "spree" for a few days and missed a small artificial denture from the front of the mouth which he thought had been lost. Roentgen examination revealed the cause of the obstruction and the plate was removed without much difficulty. The patient made an uneventful recovery.

DISCUSSION.

DR. GEORGE W. BOOT thought it was unusual for a patient to swallow a plate without knowing it and believed it was criminal for dentists to make such small artificial removable dentures. They should be large enough so that they could not slip into the esophagus.

DR. EDWIN MCGINNIS recalled a case he had seen where the patient had swallowed a plate containing three teeth. The

patient developed hoarseness and later difficulty in breathing but had no knowledge of having swallowed a foreign body. Upon examination the physician saw the upper edge of the denture and removed it with forceps.

Paper: "Tumor of the Carotid Gland With Stokes-Adams Syndrome."*

By George W. Boot, M. D.

DISCUSSION.

Dr. Otto Stein thought these tumors were probably not as common as they might seem and that they were usually diagnosed late, frequently on the operating table.

He recalled a case which he had considered a tumor of the neck of indefinite origin. Upon attempting to operate they found a dense, vascular and adherent mass, and the disturbance caused by manipulation indicated that the tumor had something to do with the pneumogastric. The effects were so pronounced that operation had to be discontinued.

Dr. Stein said he would hesitate to operate in such a case, for the mortality is enormous and the possibility of a patient living for a long time with such a gland is more favorable than that of recovery following operation. Most cases are of malignant type when seen. The location of the mass, at the bifurcation of the external and internal carotid artery so closely associated with the nerves, the hypoglossal and superior cervical plexus lying on the anterior surface, makes dissection very difficult without producing some disturbance of the nerves. In the early stages this difficulty would not be so great, but the cases are seldom seen early, and the intimate association with the carotid made them practically hopeless operations. The ability to diagnose this condition and differentiate it from many other things was of importance, for many things lead to suspicion of the carotid gland. He was of the opinion that such growths should be left alone.

Dr. Joseph C. Beck asked Dr. Boot to explain the connection between the epilepsy and the growth. He thought perhaps the relation of the sympathetic ganglion to the tumor was of some importance in this connection, along the line of the work of Jenesco regarding the ligation of the carotid to

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See page 1241.

cure epilepsy. He believed he might be as lethargic as Dr. Stein in leaving such a growth alone.

He also asked Dr. Boot if he did not find in the literature that the carotid gland was related to the polyglandular system. In his opinion, radiotherapy might have been given a trial in this case.

DR. BOOT (closing) said that another physician saw the patient and used radiotherapy before he took charge of the case. He thought Dr. Stein's advice to leave such cases alone was very good. The time to remove these growths was before the tumor got large enough to involve the carotid, but that was before he sought the advice of the physician, as a rule.

He was glad to hear Dr. Beck mention the work of Jenesco but thought it had been entirely to the discredit of the profession and believed that Jenesco himself had changed his opinion.

Dr. Boot believed the cause of the epilepsy in his case was anemia of the brain from the compression of the growth, that the sympathetic ganglion had nothing to do with it, but that it was a Stokes-Adams syndrome. They could neither feel the pulse nor hear the heart sounds with the stethoscope before the convulsion.

Paper: "Congenital Atresia of the Posterior Nares."*

By JAMES E. LEBENSOHN, M. D.

DISCUSSION.

DR. ROBERT SONNENSCHEIN said he had nothing to offer in the way of criticism or in addition to the subject matter, but congratulated Dr. Lebensohn on his clear and logical presentation of the subject and the society on the acquisition of so valuable a member.

The point regarding the lack of dependence of ear conditions upon obstruction of the nares was also emphasized at the last meeting of the Illinois State Medical Society by Dr. Shambaugh. Dr. Sonnenschein thought it important to remember that while obstructions of the nose may influence middle ear conditions when they favor extension of the infection by way of the eustachian tube, it is not necessary to

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See page 1128.

remove every little spur when operating, on the assumption that it will produce difficulty with the ear. He had recently seen a case of marked deviation of the septum to one side, but this patient had never complained of obstruction, as frequently happens. In this instance the patient asked whether the septum should be operated upon and said she had been told by another man that she must have an operation because she might get a sinus or ear infection some time in the future. As there was no obstruction Dr. Sonnenschein advised against operation.

DR. JOSEPH C. BECK said he did not know this condition was so rare and could add six cases that he had operated on in children. He offered a suggestion regarding the reclosure which the patient complained of. The electric burr is not very easily managed in the nares, even with the finger as a guide, but he believed the perforation could be very easily made by means of a large rasp which would penetrate most of the bony obstruction. If not, a hand chisel could be used for the first opening and then a rasp could be used to make the hole as large as necessary. Having completed this, a hard rubber plug, which is flattened so that it will not obstruct the nasal spaces, is drawn by means of a catheter into the nose and fastened in place by means of tape drawn into the opposite nostril with a bit of gauze over the columella. He had removed a bilateral obstruction that had existed since childhood in this manner, with good results and great improvement in speech, but ten minutes after the patient was returned to his room he was called to see him because the man was in a fit of anger because he could not talk to suit him. The nasal twang distressed him, and he insisted upon stuffing cotton into his nose so that he could get his old manner of speech back.

DR. JOHN CAVANAUGH recalled the case of a child of about twelve years in whom the parents would not permit operation. The tonsils and adenoids had been removed, the latter on three different occasions, the inferior and middle turbinates had been removed and the mother told him the doctor had said something about removing some of the ethmoids on that side. Dr. Cavanaugh discovered the postnasal obstruction in trying a through and through irrigation, but the mother would not consent to further operation.

DR. FRANK J. NOVAK, JR., asked whether there was a true anosmia, and whether Dr. Lebensohn had introduced any odoriferous substance directly into the nasal cavity. He thought that only in this way could one tell definitely whether or not there was a true anosmia.

DR. JAMES E. LEBENSOHN (closing) thought the point Dr. Sonnenschein emphasized in regard to the ear conditions being only secondarily due to nasal obstruction was important.

He was very glad Dr. Beck mentioned having such a large number of cases and felt that many cases were not reported because their rarity was not realized. He thought the profession should be more alive to this condition and thus avoid doing secondary adenoidectomies and other unindicated operations.

DR. ELLISON L. ROSS read his inaugural thesis on

Paper: "Some General Effects of Local Anesthetics Administered as in Tonsilelctomies."*

BY ELLISON L. ROSS, M. D.

DISCUSSION.

DR. OTTO STEIN asked whether there was an attempt at controls in the experiments and whether the position of the patient was taken into consideration, whether they were upright or reclining. He believed the injection of water or salt solution would produce an effect on the blood pressure.

DR. J. GORDON WILSON said that the knowledge of the synergistic action of drugs had been known for a long time. It had, for example, been recognized that two drugs together may act on a structure—e. g., the intestines with a much greater potency than the summation of the actions of each separately. The explanation is not known. But the synergistic action of adrenalin with cocain is not to be grouped with the similarity of actions of socalled adjuvants. That cocain and adrenalin together act synergistically appears to have attracted little attention from clinicians. The great importance of the consideration of such action was shown by Dr. Ross' paper and might well make one thoughtful. -

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See page 1229.

The current opinion of the conjoined action of adrenalin and cocain has been recently expressed in a textbook by a distinguished clinician, namely, that (1) by injecting adrenalin one contracts the blood vessels and stops or lessens absorption of cocain, (2) that adrenalin acts as a physiologic antidote to cocain. To the suggestion that adrenalin acts as a physiologic antidote to cocain, Dr. Ross' experiments are in contradiction. The very great rise in arterial and intracranial venous pressure from the combined action of cocain and adrenalin makes one pause and consider how little we have realized how great a disturbance of cranial circulation we have been bringing about with these so frequently and freely used drugs. It makes one pause and consider whether the advantages of swabbing a throat previous to tonsil operations in which adrenalin and novocain are injected are not offset by the disadvantages of this synergia.

Further, these experiments indicate the explanation of the symptoms frequently seen in operations following the conjoined application of cocain and adrenalin, and that we are not dealing with a diminished arterial pressure but a rise of arterial and venous pressures.

DR. EDWIN McGINNIS thought the paper was very illuminating and interesting. It bore out some clinical findings he had observed in using local anesthesia in nasal and tonsilar operations. Some years ago he eliminated the use of adrenalin in doing tonsillectomies under local anesthesia. He did not realize the synergistic action of the drugs, but used them with a definite idea in view. The reason for using the adrenalin was to reduce the absorption of the cocain and to reduce the bleeding at the time of operation. He decided that in doing operations under local anesthesia he did not care how much bleeding there was and that the adrenalin could be eliminated. He noticed that with the topical application of adrenalin in the nose he got as much upset at times as with cocain alone, and he now paints the mucous membrane once with 10 per cent cocain and then injects the novocain. In this way he gets less upset for the patient, probably because there is less synergistic action from the novocain with the elimination of the adrenalin.

DR. HARRY L. POLLOCK said he would like to see Dr. Ross experiment with the cocain application. Formerly they used the 5 to 20 per cent cocain in operating on the nose, but had a great many of the toxic effects, particularly with the weaker solution, the 5 and 10 per cent, which they attributed to the fact that they got more absorption. The higher the percentage of cocain the greater astringent action on the blood vessels and the less the absorption. For several years they had used nothing but the cocain flakes, dipped in very weak adrenalin solution and applied to the mucous membrane. Since they have followed this plan the toxic effect has been much less pronounced. In doing a suspension or in nasal work they use the cocain flakes in large amounts and have had very few untoward effects as compared with the cocain solution. He believed this was due to the astringent action on the mucous membrane and the small blood vessels preventing absorption into the system.

DR. ROBERT SONNENSCHEIN thought this was one of the most valuable papers that had been offered before the society in a number of years. Many of the statements made corroborated things which, from a clinical standpoint, he had felt for a long time. Warned by an experience in the Vienna Clinic some eighteen years ago, where two patients died immediately after a submucous injection of cocain, he had never used it. In recent years he had used no cocain at all except in those patients who were so sensitive that the introduction of the needles was very painful, or where the pharyngeal reflex was so active that it was necessary to use it. Preliminary to nose and throat operations he had seldom used morphin, because he could not see that those patients who had received morphin had any less difficulty than occurred in those cases in which it was entirely omitted but that the opiate often did cause nausea or vomiting. He thought that the simpler the technic and the less drugs that were used, the better for all concerned. The substitution of apothesin made cocain practically unnecessary for throat work. They had found very few indications of syncope or other untoward symptoms during tonsillectomy since using a modified technic with alcohol, regarding which a paper would soon be read.

Dr. George W. Boot spoke of a tonsillectomy where the operator brushed over with a 5 per cent cocain and then injected a 1 per cent novocain to which adrenalin had been added. The patient had a blood pressure of about 280 at the time of operation, but the operation was performed because pus was exuding from every crypt. During the operation the patient developed great anxiety and complained of intense pain over the region of the heart. She went into collapse, and they thought she was dead, but after giving a hypodermic of morphin the distressing symptoms passed over and the next morning she was all right. She died a year later of apoplexy.

Dr. Frank J. Novak, Jr., said that in the Army when they were unable to obtain novocain they used apothesin with good results. They had unpleasant results following the use of cocain, so they used apothesin topically for all nasal operations. While the induction of anesthesia required a longer time, the anesthesia was as complete as that obtained with cocain. Since that time he had used 5 per cent apothesin topically with excellent results in all nasal operations. He has not used cocain since 1917.

Dr. Ellison L. Ross (closing) said that their cases were in a partial reclining position when operated upon.

The psychic influence suggested as a cause for increased blood pressure was always to be taken into account. The injection of water in the place of local anesthetics had not been tried. Since a great part of the work described was done on anesthetized animals it was certain that the drugs have a very large proportion of the influence exerted.

He had not tried cocain flakes, but thought that the concentration would make no difference so long as adrenalin was injected afterward. The astringent action of the flakes of cocain would certainly tend to decrease the rate of absorption.

Dr. Ross' idea was not to condemn the use of adrenalin but of cocain. He does not use cocain at all in working on tonsils, but thought that if one wished it could be used after the injection of adrenalin and novocain. After three to five minutes the adrenalin would all be gone and so could not exert any synergistic action with cocain.

MEETING OF THE SECTION OF OTOLARYNGOL-
OGY OF THE COLLEGE OF PHYSICIANS.

PHILADELPHIA, PA., DECEMBER 20, 1922.

Extremely Large Nasal Polyp.

DR. GEORGE B. WOOD, the chairman, presented a specimen of an extremely large nasal polyp removed from a mentally defective boy of 19 years. The symptoms began during the last summer. The soft palate was pushed out to the level of the back teeth, and the polyp could be seen below the margin of the soft palate. It was removed by the following method: A rubber catheter passed along the floor of the nose was brought out of the mouth and a silk thread tied to the end of it. The thread was then brought back through the nose and tied to the middle of a wire loop. This wire was then pulled through the nose into the mouth and worked with the finger in the throat around the growth. The ends of the loop were passed through a Watson tonsil snare and the growth cut off close to its insertion under the middle turbinate, the polyp being withdrawn through the mouth.

Presentation of Two Patients With Cancer of the Nose and Throat, Treated With Radium.

BY RALPH BUTLER, M. D.

First case, J. E., widow, age 58. Was admitted to the hospital November 1, 1921, with a history of profuse watery discharge from the left nostril, which followed an attack of influenza in December, 1917. The discharge became muco-purulent and the nose was gradually blocked. No pain, no spontaneous bleeding, no loss of weight and no evidence of metastasis. Examination showed a tumor the size of a shelled walnut in the left nostril protruding into the nasopharynx and bleeds easily. Diagnosis by biopsy, papilliferous adenocarcinoma. Treatment, 25 mg. of radium in capsule was applied to the nasopharynx and 15 mg. to the left nostril for eight hours. Three weeks later the second application of radium was applied, 35 mg. to the nasopharynx and 15 mg. to the left nostril. Twenty-four days later breathing seemed a little

easier and the same dose of radium was used. This was repeated twenty-seven days later and then no treatment for two months. On March 23, 1922, 50 mg. of radium was applied for twelve hours. April 11, 1922, 35 mg. was applied into the left nasal cavity and was repeated one month later for eight hours. This time the mass in the nose seemed to be decreasing in size and the patient felt better. June 1, 1922, the patient complained of a purulent discharge from the right nostril since the last treatment. Radium was again applied to the nose June 22, 1922, 50 mg. for eight hours. Condition improved.

Second case.—S. S., male, age 54. Was admitted to the hospital November 26, 1919, with the following history: Two years previous patient noticed a deafness in the left ear, which continued to progress. No pain. On examination, an ulcerated area was seen in the nasopharynx. Physical examination negative. No metastasis, no loss of weight, blood Wassermann negative. December 11, 1919, 35 mg. of radium, in capsule, was applied for twelve hours. Six weeks later same treatment was repeated. No unusual effects except dryness of mouth and throat. March 18th, examination showed improvement, decrease in size of the growth and gain in weight. Radium was again applied September 15, 1921. Fifteen mg. applied twelve hours, March 21, 1922. April 11, 1922, reduction in the size of the growth but there was evidence of radium burn. April 27, 1922, patient had evidence of radium burn in the nasopharynx and a perforation of the soft palate. Radium treatment was applied September 14, 1922.

DISCUSSION.

DR. GEORGE B. WOOD stated that he had seen one case of squamous cell epithelioma, about the size of a half dollar, located on the posterior pharyngeal wall, completely disappear after one week's treatment with radium. He had seen the patient today, two weeks after the treatment, and only a little whiteness of the mucous membrane, resembling a leukoplakia, could be seen.

DR. GEORGE M. COATES stated that he saw a case of sarcoma involving the soft palate and lateral pharyngeal wall in 1916.

It was incised and radium inserted into the opening, with the result that the growth disappeared, just as in Dr. Wood's case, within a week. No return two months later. After a two weeks' business trip, the patient returned with the growth just as it was in the beginning, and it went on to the termination.

Dr. Fielding O. Lewis spoke in favor of the Crosby Green operation when the malignancy involves the ethmoid or maxillary sinuses or both. It makes a very desirable means of applying radium to these regions. This method has been used in the radiologic department of the Philadelphia General Hospital. After the surgical procedure, bare tubes are introduced into the involved structure, and radium plaques are applied outside. The results have been most gratifying.

Report of a Case of Brain Abscess of Otitic Origin With Exhibition of Brain.

By George M. Coates, M. D.

The patient was a middle aged man, with a history of a running ear on the left side since childhood, but had not had any other symptoms until now. When first seen he had been ill for two weeks. Examination showed the external auditory canal was swollen closed, no particular signs of mastoiditis. He complained of vertigo and deafness. Operation revealed a noncellular type of mastoid, with cholesteatoma in the mastoid antrum and a small cell adjoining. The radical operation was without incident, and the patient was discharged from the hospital in ten days in apparently good condition. Fourteen days following the operation he developed a facial paralysis, no vertigo. Barany tests showed no response in the vestibular apparatus on the affected side. Secondary operation five weeks later showed that the labyrinth was necrosed, and the anterior wall was removed in one large sequestra. The whole petrous portion of the temporal bone was found destroyed. The patient had complained of severe headaches on the left side before the last operation. Brain abscess was suspected, but the marked destruction of the temporal bone was thought sufficient to account for the headaches. Paralysis of the tongue and pharynx developed on the tenth day following the

operation, and death occurred a few days later. On necropsy there was a diffuse, purulent meningitis covering the entire base of the brain. The petrous portion of the temporal bone was destroyed. The pyramid looked normal, but it was found so thin that a knife would puncture it at any point, going immediately into foul smelling bone. On a cross section of the brain, three abscess cavities were found in the temporo-sphenoidal lobe, one near the cortex. The whole left side of the brain was softened and flattened out.

DISCUSSION.

Dr. George B. Wood.   A man in the army was shot in the ear about a year before he came under my observation. He was admitted to the Pennsylvania Hospital with evidence of intracranial pressure. The bullet was found to be lying in front of the petrous portion of the temporal bone on the level with the posterior internal wall of the tympanic cavity. The patient developed a series of brain abscesses, and later a hernia of the brain as large as a hen's egg, death following three months later. His whole frontal lobe was gone; there was scarcely any brain substance in it. The parietal lobe was in the some condition, as was also the occipital lobe. Practically one-half of the man's brain was gone, and yet he lived for three months. He was able to eat and attend to normal functions of life until a few days before he died.

Dr. Walter Roberts stated that the cases he had seen were similar to the one that Dr. Coates reported, but usually they had been tubercular in origin. He mentioned a conversation with Dr. Lewis, wherein there was a brain abscess in the frontal lobe secondary to frontal sinus disease, in which there were a few symptoms showing that a great destruction of brain substance can occur without symptoms.

Dr. Fielding O. Lewis: The case mentioned by Dr. Roberts was a young man, who had a history of long continued purulent nasal discharge due to frontal sinus disease. Radical operation was performed. Convalescence was uneventful until three weeks following the operation, when he developed signs of pus in the frontal regions, swelling, redness and fluctuating of the skin, later a secondary operation. After exposing the

whole region of the frontal area, a small necrotic portion was found in the posterior plate of the left frontal sinus. On curetting this area, as much as two ounces of pus were evacuated from the brain. The patient had not had any eye or intracranial symptoms. He at this time is in splendid condition. No rise in temperature, mentally clear and feels good. The cultures from the pus showed streptococci and staphylococci.

Dr. S. MacCuen Smith stated that one of the most interesting points about intracranial abscesses, otitic in origin, involving the silent area, was the absence of symptoms. In his experience, about one-half of the brain abscess cases were discovered during the radical operation for the cure of chronic suppurative middle ear disease or mastoid disease. Before the advent of the epidemic of influenza his brain abscess cases were chiefly the result of the chronic suppuration form of middle ear disease. Since the influenza epidemic the largest number of brain abscess cases have been of the acute type. When there are symptoms of brain abscess and it is not possible to determine its location, his experience had been that inasmuch as the vast majority of all brain abscesses of otitic origin occur in the temporosphenoidal lobe one is justified in exploring the temporosphenoidal lobe before seeking for the abscess in any other locality. On operating for brain abscess he invariably follows the route of infection. He has never had a case live following an evacuation of pus from any part of the brain unless this method was followed. He was interested to see in Dr. Coates' specimen that there were several distinct abscess cavities. He does not share the German view that there is always a communication between the various cavities even though they may not be found.

Dr. Fetterolf: Several years ago Professor Behrens of New York reported a case of brain abscess and rather emphasized that you so often have them without symptoms. It was brought out in the discussion, and rather stressed by several men, that a careful study and search about the disposition of the individual would often show that some changes, in connection with other things, would tend to point out the fact that a brain abscess might be present. There is often a change in the psychology of the patient that might suggest a brain abscess.

Dr. Ralph Butler wished to emphasize the point made by Dr. Fetterolf by citing a case which he had some years ago at the Lankenau Hospital. A man had been shot through the upper jaw and the ball was removed through the orbit. Nothing abnormal was suspected in the brain. He became very objectional in the ward, caused the nurses a great deal of trouble and his whole disposition was changed. An abscess of the brain was later found.

Dr. S. MacCuen Smith brought up the question of aphasia, especially what might be called optic aphasia, and related a case at the Jefferson Hospital, on whom he had operated for brain abscess. The patient had a particular type of aphasia. When he was shown a key he could not tell that it was a key. He would point to the door and show that it was meant to open the door. When shown a knife, would show that a knife was meant to cut something. He was of the opinion that optical aphasia is as important a symptom as a changed disposition.

Dr. Coates, in closing, stated that his patient had shown both a changed disposition and aphasia. He thought that the abscesses shown in the brain were of an acute type because they were totally without capsule. The final diagnosis was a temporosphenoidal abscess, but at that time, in view of the extensive destruction of the petrous pyramid, and on account of his very poor condition, he did not deem it advisable to search for the abscess. His experience corresponds with that of Dr. Smith in that they should be opened through the line of infection.

Paper: "Congenital Atresia of Nostril."

By Warren B. Davis, M. D.

Female patient, born December 20, 1920, full term. Family history negative, excepting that the patient is one of twins. Normal in all respects, excepting atresia of right nostril. There was a shallow dimple where the right anterior naris should have been, only partial development of the right alar cartilage and more depression of the bridge of the nose than could be considered normal. Most probable cause of the malformation

was some continuous pressure on the lateral nasal area during early fetal life by contact with the other twin.

When the patient was five months old double "Y" incisions were made in the slight depression where the nostril should have been located. The skin flaps thus formed were separated from the underlying tissue. Incisions were made through the soft tissue, keeping the scalpel parallel with the nasal septum as it was carried posteriorly. The atresia was found to extend to approximately a coronal plane at the junction of the anterior and middle thirds of the lower turbinate. The opening was enlarged by the use of a nasal dilator. The skin flaps were placed over the anterior margins of the nostril and a small perforated hard rubber splint placed in the nostril and held in position by a silkworm gut suture passed through the floor of the naris and tied underneath the upper lip. Previous to operation epiphora had been continuous. There was marked improvement within three days after operation. Contraction of scar tissue after operation was difficult to control in spite of persistent treatment by special dilators. Secondary operation will be required in later childhood. It is believed that the ventilation secured by the formation of the small nostril will allow proper development of the nasal accessory sinuses and proper function of the nasolacrimal duct.

DISCUSSION.

DR. BENJ. D. PARISH stated that he had seen, about a year ago, an interesting case of congenital atresia of one nostril in a child two weeks old. The right nostril was entirely absent with the exception of a little tab of skin, which was found patulous coming from the inner canthus of the right eye. On probing, it was found to be apparently the lacrimal duct opening directly into this tube. The question arose whether there was any posterior opening under the posterior superior space and what might be done. The parents were quite willing and anxious to improve the cosmetic appearance, so the lacrimal duct was split and an incision made along the bridge of the nose and another incision along the cheek in order to use this flap for the right nostril. The result looked well following the operation, but as it was not his own case he was unable to tell what the final result was.

A Radical Operation, Three Years Ago, for Adenocarcinoma of the Ethmoid, Antrum and Middle Turbinate—No Recurrence. (Presentation of Patient.)

By DAVID HUSIK, M. D.

(By invitation.)

In reviewing the literature of carcinoma of the inner nose and accessory sinuses he found that the authoritative opinion was that this condition is hopeless. Bloodgood has stated that not one of eighteen cases with malignant disease of the nares operated upon at the Johns Hopkins Hospital recovered and that there is no authentic case on record where recurrence has not taken place before the patient has left the hospital. Intranasal carcinoma exists in several forms. The order of the frequency is, 1, squamous celled; 2, cylindrical celled; 3, medullary celled, and, 4, adenocarcinoma. Alexander reporting on adenocarcinoma of the nares found one case aged 22 years, one aged 28 years, and one aged 33 years. The author's case is 32 years old. In 200 reported cases of intranasal carcinoma only 21 were undoubted cases of adenocarcinoma.

Jonathan Wright states that he has never seen in the nose and throat, where ablations of various growths are often made at repeated and long extended intervals, any evidence of a histologic change in type from the benign to the malignant in either papillomatous or adenomatous growths. This view is not held by others, who believe that adenomata and papillomata frequently become carcinomatous, and they account for the rarity of adenocarcinoma as being due to the rarity of intranasal adenomata.

The author reports the following case for the reason that the patient is free from recurrence three years after operation for removal of an adenocarcinoma involving the middle turbinate, antrum and ethmoid on the left side: A. W., age 32, reported to the University of Pennsylvania Hospital in May, 1919, complaining of left sided nasal obstruction and discharge. Examination showed the left nostril filled with a large grayish irregular polypoid mass, covered with a mucopurulent discharge, boggy to touch, and evidently attached to the middle turbinate. Transillumination showed a dark area below the

left eye and over two-thirds of the internal aspect of the antrum. Under cocain adrenalin solution enough of the growth was removed to establish nasal breathing. The patient disappeared but returned to the hospital November 2, 1919, with a severe left sided nasal hemorrhage. Examination at this time showed the left cheek and eye more prominent, and he had developed left sided deafness. The following biopsy study by Dr. Allan J. Smith was given: "The general appearance of the histologic structure would tend to its classification as an infiltrative adenocarcinoma of origin in a mucous secreting gland, such as occur in the mucous membrane of the nose." X-ray report showed disease of the antrum, anterior and posterior ethmoids. Fifty mg. radium in capsule was directly applied to the growth every seven days, and X-ray treatment was given by Dr. Pancoast with little effect. On December 18, 1919, radical operation for removal of the upper jaw was performed and the ethmoids were curetted.

Several days following the operation an acute otitis media developed which promptly cleared up. In March, 1920, there was seen evidence of recurrence in the region of the superior turbinate, which disappeared after applying 50 mg. of radium. The biopsy report was adenocarcinoma.

<center>DISCUSSION.</center>

Dr. Fielding O. Lewis stated that Dr. Husik should be congratulated upon the splendid result, and he thought that the use of radium and X-ray was very timely used.

Dr. George M. Coates: Dr. Husik, I think, quoted Dr. Bloodgood to the effect that these cases never recover. The Boston physicians have shown that they do recover. Their plan is the open face method, keeping the antrum open until the possibility and probability of recurrence is over.

<center>Laryngeal Gumma—Ten Year Old Child.

By Joseph D. Seiberling, M. D.

(By invitation.)</center>

Evasive symptoms, the etiology of which is obscure, should direct one's attention to the probable factor being syphilis.

This is true especially just preceding or during adolescence. I could do no better than to refer to Hunter's Commentary on Syphilis in which he states, concerning laws of evolution of hereditary syphilis, "It is certain that an infant may pass through the secondary stage of inherited syphilis without ever presenting any symptoms. Those who have shown no symptoms in infancy may yet suffer in later life. It is clearly to be understood, however, that where this happens to adolescents, they suffer from a class of symptoms wholly different from those of infancy. It is not that the secondary stage has been delayed, but simply that it has passed through without ostensible disturbance. After a period of latency there will in many cases come a group of very peculiar affections, involving principally the sense organs."

D. S., 10 years of age, Russian parentage, born in the United States. Good health since birth. One uncle died of tuberculosis and another of cancer. Father 48, living and well. Served in the Russian Army. Two years ago had swelling of the thumb, which disappeared under mixed treatment. No Wassermann blood test at that time. Denies any knowledge of visible lesion. Has had occasional headaches, also fullness in the head. Blood Wassermann plus two. Mother 47, living. Had 13 pregnancies. The fifth, sixth and eleventh terminated at ten weeks, three and one-half and six months, respectively. First child died of hemorrhage. Third child died three weeks after birth from infection following circumcision. Eighth child died of scarlet fever at three years of age. Ninth child died of diphtheria at one year. Six children living and well. The subject of this report is the twelfth child. History of present illness: Six months ago, after returning home from school, noticed hoarseness, no sore throat. The hoarseness progressed until the voice became only a whisper. Was advised by family physician to have tonsillectomy. Reported to the Polyclinic Hospital in September, 1922. General physical examination negative. X-ray examination showed no metastasis in the lungs. Blood count shows: Erythrocytes, 4,610,000; leukocytes, 9,300; color index, 90. Blood Wassermann positive, plus four. Examination of the larynx showed left ventricular band and cord

infiltrated with neoplasm, which projected beyond the median line, somewhat irregular but smooth in contour, seminodular slightly and apparently fixing the cricoarytenoid joint. Right vocal cord slightly congested but otherwise normal and moves freely. Glottic chink narrowed to about one-third normal. Slight retraction of suprasternal notch on inspiration. September 21st, received 0.45 arsphenamin intravenously and this treatment has been continued once weekly up to the present time. Retrogression of the mass has been rapid.

DISCUSSION.

DR. GEORGE B. WOOD: This is an extremely interesting case, gumma in children being very rare, and especially gumma of the larynx.

DR. WALTER ROBERTS stated that in his experience the blood Wassermann reaction is often useless as a diagnostic measure. He has used potassium iodid as a therapeutic test in the diagnosis and was of the opinion that good results are often obtained when little anticipated by the use of ascending doses of potassium iodid, althouglr repeated Wassermanns have been negative.

DR. CURTIS C. EVES stated that Dr. Roberts' remarks called to mind a case he had treated some time ago. A man 70 years old with neoplasm of the larynx. Three negative blood Wassermanns. No specimen had been removed for diagnosis. Patient was treated with radium and X-ray with little improvement. Later the patient's family physician administered potassium iodid for some asthmatic symptoms and the growth began at once to disappear.

DR. SIEBERLING: The patient's Wassermann is still plus four.

Paper: "A Case of Cerebrospinal Rhinorrhea."

BY JOHN EDWARD LOFTUS, M. D.

(By invitation.)

The author, after giving a complete review of all published cases and literature of cerebrospinal rhinorrhea, the compre-

hensive and thorough data as to the etiology, pathology, symptoms and diagnosis, reports the following case: A woman aged 40, was first seen January 5, 1922, after being treated three months for sinusitis. She was complaining of a constant dripping of clear fluid from the left nostril for the past three months. The patient has always been very stout. Present weight, 240 pounds. No history of any head injury. Present condition began October 23, 1921, after a very hearty laughing attack, which was followed by severe sneezing, patient becoming aware of a clear limpid fluid from the left nostril associated with a frontal headache extending to the vertex. The headache has been constantly present but diminishes somewhat when the flow increases. One-half an ounce of fluid escaped in one hour. The fluid was clear, odorless, tasteless and without sediment, alkalin reaction. The flow of the fluid is greatly altered or increased by the position of the head. When the head is dropped forward, the flow increases; when the head is ·in the upright position, the flow is less; when lying in bed with the head hanging over the edge, face downward, there is a drop of fluid every two seconds. During sleeping hours the flow is the same as during the day. The constant swallowing of the fluid at times has caused severe diarrhea. On three occasions the patient states that the fluid escaped from her left ear, that it came out in gushes and lasted a short time. This was not seen by the author. The fluid is also increased when the patient works, climbs steps, does any severe exercise or when she becomes angered and excited. Examination of her nose revealed a sharply deviated septum to the right. There is a spur at the base of the septum on the same side which touches the inferior turbinate. Tonsils are of average size and apparently normal. The sinuses were negative. The author was unable to determine definitely where the fluid made its exist but was of the opinion that it came from the posterior portion of the roof of the nose and not from the middle meatus. Examination of the ear was negative. Wassermann reaction negative. Examination showed that the escaped fluid had the same composition as that of a normal cerebrospinal fluid. Eye examination had no bearing on the symptoms. Complete neurologic examination showed no evidence of organic disease of the brain.

Conclusion.—"Patient became so depressed and melancholic that it was deemed advisable to send her to the hospital for a rest cure. After being in the hospital two and a half months the flow of fluid from her left nostril entirely subsided; two weeks following the cessation of the flow she was suddenly taken with a chill and a rise of temperature of 104°, associated with this was numbness of the entire body and severe headache. Two days later she went into a semicomatose state with alternate periods of lucidity. The following day she developed a facial paralysis and tremors of the entire body. Two days following she developed auditory nerve deafness and three weeks later died of acute encephalitis.

DISCUSSION.

DR. FETTEROLF: To my mind, this was one of the most notable presentations to which I have ever listened, for carefulness of study and for accuracy and clearness of presentation it is particularly notable.

DR. WARREN B. DAVIS stated that he had been asked to see the patient while in the St. Agnes Hospital because of her pain, which had developed in the left ear. He stated that examination of the ear was negative. There was one point, which he stated Dr. Loftus had not mentioned, and that was the possibility of a fracture having been made at the time of a submucous resection, which had been performed some time before, but was unable to determine whether this was the case. The upper part of the resection seemed to have been done fairly high. There was a firmness of the septum where Dr. Loftus said there was still a ridge. There was also a slight palsy on the left side at the time of his examination.

DR. GEORGE B. WOOD inquired if a postmortem had been secured.

DR. LOFTUS stated that unfortunately, due to the absence of a pathologist, a postmortem had not been done. He had been given the privilege, but the proper person was not around at the desired time. Regarding the escape of cerebrospinal fluid from the ear: In reviewing the literature, he found that Escat reported a case of cerebrospinal fluid from the left ear following a submucous resection. No other cases were found in the literature.

A Series of Colored Paintings Illustrating in Progression the Pathologic Conditions of the Membrana Tympani.

BY JAMES H. MENDAL, M. D.

(By invitation.)

The author showed in succession painted illustrations 200 times larger than the normal size of the drumhead, taking up in succession the normal ear drum and the various pathologic conditions which are found. The plates represented an enormous amount of work and careful study made by the author. The minute details that were brought out and explained were both interesting and instructive to all members present.